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POSITIVE ROENTGENOGRAMS PLUS POSITIVE SPUTUM SMEARS DO NOT ALWAYS EQUAL PULMONARY TUBERCULOSIS*

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FOR several years bacteriologists and physicians specializing in the treatment of tuberculosis have been reporting instances in which acid-fast organisms which were not *Mycobacterium tuberculosis* have been associated with pulmonary disease in human beings.[1–9] These microorganisms cannot be distinguished by means of microscopic examination of stained smears of sputum, nor can the disease process in the lungs be distinguished from tuberculosis by means of roentgenologic examination.

It is our purpose to report 7 cases in which such a combination of facts existed in order to emphasize again that the roentgenologic diagnosis of pulmonary tuberculosis is presumptive and should be confirmed by identification of the specific microorganism by means of its cultural characteristics and virulence for laboratory animals. We wish to emphasize also that the presence of acid-fast bacilli in a stained smear of sputum does not necessarily confirm a roentgenologic impression of pulmonary tuberculosis. In other words, "a positive roentgenogram plus a positive sputum smear does not always equal pulmonary tuberculosis."

REPORT OF CASES

CASE 1. A married white woman, forty-five years of age, was first examined at the Mayo Clinic on November 15, 1948. She complained of dizziness and of attacks of pain in the epigastrium which were sometimes associated with nausea and vomiting. These symptoms had been present for about five months. Her only respiratory symptom was a mild cough, which she noticed only in the morning and which produced a small amount of mucoid sputum. Nothing abnormal was found at the time of physical examination and the examining physician's provisional diagnosis was Ménière's syndrome.

Unexpectedly, stereoscopic roentgenograms of the thorax disclosed evidence of disease in the upper lobe of the left lung (Fig. 1). Cavities were present in the diseased segment and the pleura was thick in this region. The roentgeno-

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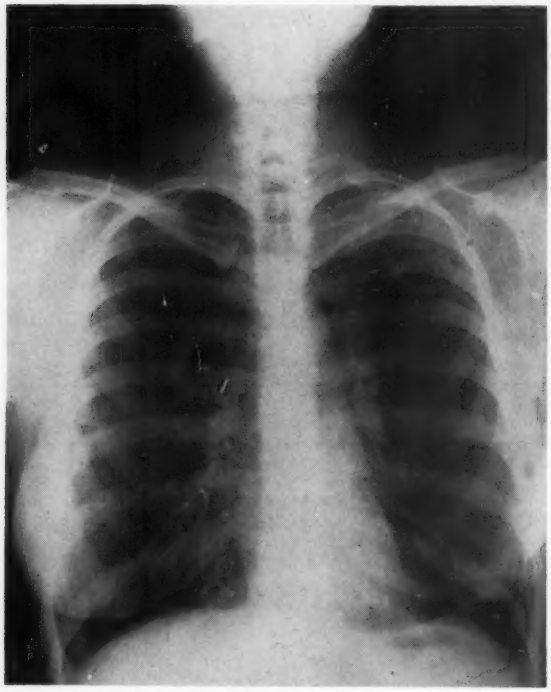

FIG. 1. Case 1. Large cavity in upper lobe of left lung discovered by means of roentgenogram made routinely as part of a general examination. Patient had no respiratory symptoms.

logic impression was that of active tuberculosis with cavitation.

A specimen of sputum was obtained from the patient and many acid-fast bacilli were seen in the stained smear. Since this finding seemed to confirm the roentgenologic findings, the patient was sent to a sanatorium for treatment. At the same time, cultures of the sputum were begun and some of the material was injected into guinea pigs. Many specimens of sputum were examined and in each instance acid-fast bacilli were seen in great numbers.

The first inkling that all was not what it seemed came when the bacteriologist reported that the only organism that grew in the original cultures was definitely not *Mycobacterium tuberculosis*. It produced yellowish-mucoid colonies which were not typical for the tubercle bacillus. Subsequently, 2 guinea pigs which had been inoculated with sputum were killed, but on examination they were not found to show evidence of tuberculosis.

When these results were communicated to the physician in charge of the patient at the sanatorium, new specimens of sputum were obtained, new cultures were prepared and new guinea pigs were inoculated. In every instance the results were the same: an acid-fast bacillus developed which produced yellowish-mucoid colonies and which did not kill guinea pigs.

The patient was eventually dismissed from the sanatorium still feeling rather well and with the knowledge that her pulmonary disease, which showed roentgenologic evidence of improvement, was not tuberculosis. She was last seen on April 14, 1952, at which time she stated that she was in good health. Unfortunately, a roentgenologic examination of the thorax was not obtained at that time.

Comment. This is a good example of the discovery of pulmonary disease by means of a roentgenogram made routinely as a part of a general examination. It also illustrates the value of obtaining cultures and virulence tests in any case in which acid-fast bacilli are found in stained smears of sputum. It cannot be proved from the available data that the microorganism which was recovered many times from the patient's sputum was the cause of her disease.

CASE II. A married white woman, thirty-three years of age, was examined at the Mayo Clinic on April 22, 1947. She stated that in January, 1946, during the course of an annual physical examination, a roentgenogram of the thorax was said to show active tuberculosis in both apices. Subsequently, smears of her sputum were examined and reported to show many acid-fast bacilli. She followed a regimen of rest in bed for several months and was told in October, 1946, that the so-called spots were healed. An examination in March, 1947, disclosed reactivation of the lesions and, again, acid-fast bacilli in the sputum.

During all of this time the patient had experienced few respiratory symptoms and generally had felt well. She coughed occasionally but had never experienced pain in the thorax, fever, loss of weight, or hemoptysis.

The only abnormality found at the time of physical examination was a tender mass partially filling the pelvic cul-de-sac on the right side. It was thought to be a cyst of the right ovary and to be unrelated to the pulmonary disease.

A roentgenogram of the thorax showed a cavity in the upper lobe of the right lung. Many acid-fast bacilli were found in smears of the sputum. Cultures were begun, and sanatorium care was recommended. Three weeks later the

bacteriologist reported that the only microorganisms to grow in the cultures produced yellowish colonies; they were acid-fast in stained smears but were definitely not *Mycobacterium tuberculosis*. The patient was advised to return for more intensive study.

On October 1, 1947, the patient was re-examined. She had not lost weight during the interval of six months and had felt quite well. Her only respiratory symptom was cough which was occasionally productive. Results of a tuberculin skin test were strongly positive at this time. Bronchoscopic examination was carried out and material was gathered separately from the right and left branches of the bronchial tree. Stained smears of this material disclosed acid-fast bacilli. Cultures begun at this time produced yellowish colonies of a microorganism which the bacteriologist said was not *Mycobacterium tuberculosis*. Guinea pigs inoculated with the material obtained at the time of bronchoscopy did not die. They were killed and examined, but evidence of tuberculosis was not found.

Subsequent examinations in 1948, 1950 and 1951, together with written reports from her home physician, indicated that, although the patient continued to feel well, the roentgenologic evidence of her disease was increasing (Fig. 2*a*). Once in 1950 she had coughed up blood-streaked sputum. During these years many specimens of sputum were examined by means of stained smears, cultures and inoculation into guinea pigs. In every instance the results were the same: the microorganism was not *Mycobacterium tuberculosis;* it was the same chromogenic acid-fast bacillus that had been found originally.

When it became apparent that the cavity in the patient's lung was enlarging progressively, surgical treatment was recommended. The patient finally consented and on October 16, 1956, after several weeks of treatment with streptomycin and isoniazid, thoracotomy was carried out. A roentgenogram obtained a few days before operation is shown in Figure 2*b*. At operation it was found that the upper lobe of the right lung was contracted, inflamed and almost replaced by a large cavity. This lobe was removed, together with a portion of the superior segment of the right lower lobe which was adherent to it. Several firm nodules, which were thought to represent granulomas, were felt in the remaining portion of the right lung. After examining the excised pulmonary tissue the pathologist made a diagnosis of chronic granuloma with cavitation. A portion of the tissue was studied bacteriologically by means of culture and injection into guinea pigs. A chromogenic acid-fast bacillus was obtained which was not virulent for the guinea pigs.

The patient was last examined October 28, 1957 (Fig. 2*c*). She felt well, was rid of her cough and had gained about 10 pounds. No organisms were found in stained smears of sputum.

Comment. In this case the organism which was present in the sputum subsequently was shown to be present in the diseased portion of the lung. Surgical treatment resulted in an apparent cure. It seems quite likely that the chromogenic acid-fast bacillus was the cause of this patient's pulmonary disease. An extensive bacteriologic search was made for other organisms which might have caused the disease, but none were found.

Case III. A white man, sixty-two years of age, was examined at the Mayo Clinic on September 19, 1949. About nine months before, he had experienced a chill followed by high fever, with one recorded temperature of 104° F. A cough developed, productive of yellow sputum, with pain of a pleuritic nature in the right side of the thorax. The physician who saw him made a diagnosis of pneumonia and treated him with penicillin given intramuscularly. Two or three days after the onset of his illness a roentgenogram of the thorax was made and a sample of sputum was obtained for staining and microscopic examination. The patient was told that the right lung showed evidence of disease and that the sputum was "positive." He was advised to enter a sanatorium for collapse of the lung in treatment for tuberculosis, but he refused. Six weeks later he consulted a different physician who, without roentgenologic examination or study of the sputum, told the patient to stop worrying and to continue his normal routine of living. At this time he had no fever or cough. In August, 1949, he again sought medical advice; again he was told that he was well in spite of the fact that no roentgenograms were obtained. He came to the clinic feeling well but wishing to be reassured.

At this time he had no respiratory symptoms except that he coughed up a little sputum early each morning. He attributed this to the fact that he smoked a great deal. On physical examination it was found that the breath sounds were decreased and distant bilaterally. The

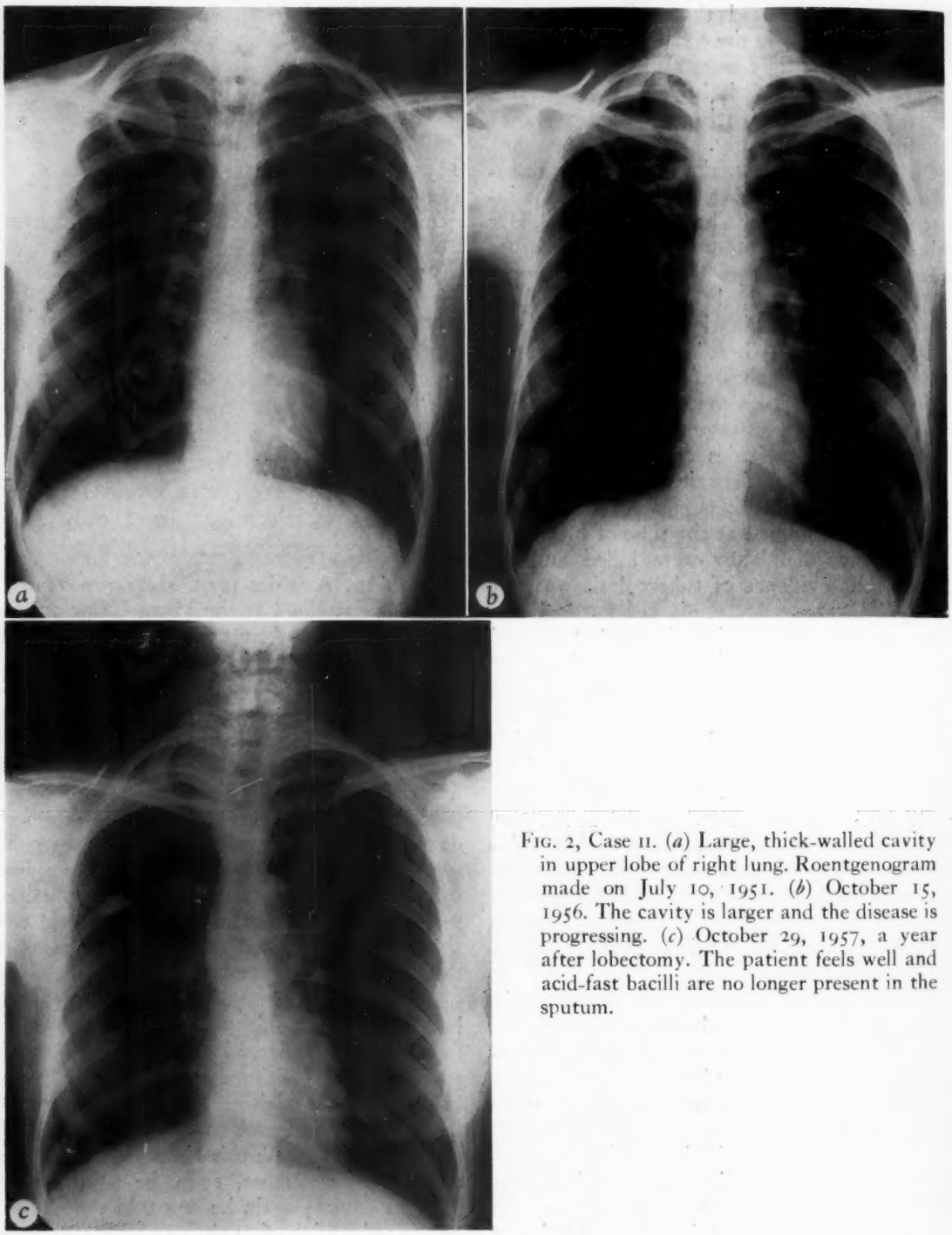

FIG. 2, Case II. (*a*) Large, thick-walled cavity in upper lobe of right lung. Roentgenogram made on July 10, 1951. (*b*) October 15, 1956. The cavity is larger and the disease is progressing. (*c*) October 29, 1957, a year after lobectomy. The patient feels well and acid-fast bacilli are no longer present in the sputum.

percussion note was hyperresonant over most of the thorax. No rales were heard.

A roentgenogram of the thorax (Fig. 3) was reported to show evidence of cavitating tuberculosis in the upper portion of the right lung. Acid-fast bacilli were found in the sputum on microscopic examination. A tuberculin skin test gave positive results. Acid-fast bacilli were cultured from the sputum, but when these were injected into guinea pigs the animals did not die. After the animals were killed examination failed to reveal any evidence of disease. The bacteriologist reported that, although the cultures continued to look like those of *Mycobac-*

terium tuberculosis, another guinea pig inoculation gave negative results.

No follow-up information is available.

Comment. This case is not so well documented as Cases I and II. However, it is particularly important since the roentgenogram of the thorax showed typical evidence of tuberculosis; the tuberculin test gave positive results; the sputum smear revealed acid-fast bacilli, and the cultures of the sputum produced colonies that looked like *Mycobacterium tuberculosis*. Only the inoculation of guinea pigs revealed that this patient did not have pulmonary disease due to *Mycobacterium tuberculosis*.

Case IV. A white man, fifty-six years of age, was first examined at the Mayo Clinic on November 23, 1951. He stated that he had been well until three years previously, at which time he had spent three weeks in bed because of an attack of influenza which was characterized by coryza, cough, fever and generalized malaise. During the ensuing six months he had lost 50 pounds and had experienced frequent headaches, fever and pain between the shoulder blades. Although he had been able to work, he had not felt entirely well and finally, in February, 1951, he had consulted a physician. A pulmonary lesion was evident in a roentgenogram and he was sent to a sanatorium. Treatment included the use of streptomycin and para-aminosalicylic acid; pneumoperitoneum was performed. At this time acid-fast bacilli were found in the sputum. On culture, these produced yellow colonies. Direct inoculation of sputum into guinea pigs failed to produce any disease, while inoculation of the cultured microorganisms did not produce lesions in the viscera but caused caseous lesions localized to the site of subcutaneous inoculation. After eight months of treatment in the sanatorium, the patient was much improved and was dismissed.

Fig. 3. Case III. Roentgenologic diagnosis, "cavitating tuberculosis, right upper lobe."

When he presented himself for examination, he complained of burning substernal pain which was unrelated to exercise or respiration. Although prior to his treatment in the sanatorium his cough had been unproductive, he now raised approximately 1 ounce of yellow-green sputum daily. A few rhonchi were heard anteriorly over the left upper portion of the thorax, and one examiner reported cavernous breath sounds in this region. A cavitating lesion in the upper lobe of the left lung was disclosed by roentgenograms made at this time (Fig. 4).

A tuberculin skin test gave positive results as did the histoplasmin test. A concentrated specimen of sputum contained many acid-fast bacilli. Cultures of this specimen of sputum produced only yellow colonies of acid-fast bacilli. No organism grew from cultures of aspirated contents of the stomach. Guinea pigs inoculated with sputum did not die; when killed and examined, they did not show any tuberculous lesions.

The left upper lobe of the lung was removed on December 1, 1951. The pathologist described the resected specimen as one showing evidence of fibrocaseous tuberculosis with multiple small cavities. No microorganisms could be seen on microscopic examination of this tissue. Unfortunately, cultures of the resected lobe were omitted.

The patient returned for observation and treatment on five subsequent occasions. In 1953 a bronchopleural fistula had developed secondary to the insertion of a polyvinyl-formal (ivalon) prosthesis, which had been used to fill the dead space in the left thoracic cavity after removal of the upper lobe. Eventually another thoracotomy was necessary to remove the prosthesis, after which the fistula closed and the patient had no further trouble. At each visit, cultures of material aspirated from the stomach

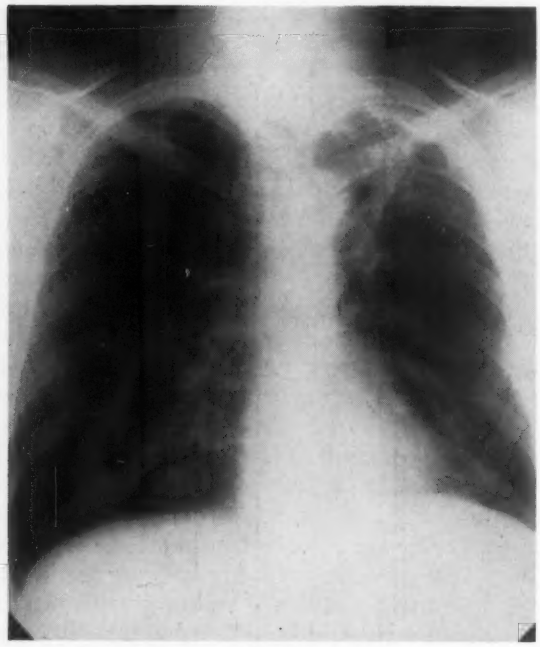

FIG. 4. Case IV. The roentgenologic appearance cannot be distinguished from that of pulmonary tuberculosis. There are many small cavities.

were prepared, but at no time did they produce *Mycobacterium tuberculosis*.

The patient's last visit was in April, 1957. At this time he had no complaints referable to the thorax. His principal trouble at this time was a gastric ulcer for which partial gastrectomy was done on April 11, 1957. Subsequent to the operation, atelectasis of the lower lobe o the left lung developed and bronchoscopic aspiration was performed. His condition improved temporarily but he died on April 21, 1957. Necropsy showed involvement of the right lung and all of the remaining portion of the left lung by bronchopneumonia. A large emphysematous bleb was also found in the upper lobe of the right lung. Neither microscopic examination nor culture of pulmonary tissue showed evidence of tuberculosis.

Comment. In this case both surgical resection and subsequent necropsy failed to provide bacteriologic evidence of tuberculosis. Although evidence that the pulmonary disease was caused by chromogenic acid-fast organisms is not unequivocal, nevertheless, such a conclusion is quite tenable.

CASE V. A white sixty-eight year old physician requested an examination at the clinic because he had lost 20 pounds over a three year period and had coughed up blood-streaked sputum. Several months before, he had been examined by a physician who specialized in the treatment of tuberculosis. The results of this examination, which included roentgenograms of the thorax, bronchograms, bronchoscopy, and cytologic as well as bacteriologic study of the sputum, were reported to be negative.

Our examination disclosed an asthenic person who gave evidence of recent loss of weight. There was no other significant physical finding. Roentgenograms of the thorax, which included laminagrams (Fig. 5, *a* and *b*), disclosed evidence of disease in the apex of the right lung with numerous small cavities. Acid-fast bacilli were demonstrated in two specimens of sputum, and an organism which was not *Mycobacterium tuberculosis* was grown in cultures of this material. Six guinea pigs were inoculated, some with sputum and some with material from the cultures, but none died, nor did any show evidence of disease when killed and examined.

The patient was re-examined recently, about four years after his first visit to the clinic. He had been feeling well and his weight had increased from 130 to 142 pounds. His cough was not so severe as it had been and the sputum no longer was streaked with blood. Roentgenologic examination showed that the pulmonary lesion had improved, and examination of the sputum failed to disclose acid-fast bacilli in the stained smear. Cultures of the sputum and gastric content have been started, but no report is available at the time of this writing.

Comment. Although this case is incompletely documented, it is probable that it deserves to be included in this group.

CASE VI. A married white woman, fifty-two years of age, came to the Mayo Clinic on October 15, 1955, complaining of cough which had begun about sixteen years before. She gave a history of three attacks of pneumonia in 1926, 1938 and 1939. Cough had developed after the last attack of pneumonia and had been productive of three teaspoonfuls of clear watery sputum each day. She had never coughed up blood. The cough was associated with a low-grade fever with temperatures up to 99° F. each afternoon. She tired easily.

She had previously consulted a physician who had made a roentgenogram of the thorax but had not found any abnormality. In 1945 she was hospitalized for ten days, but no diagnosis was made insofar as she knew.

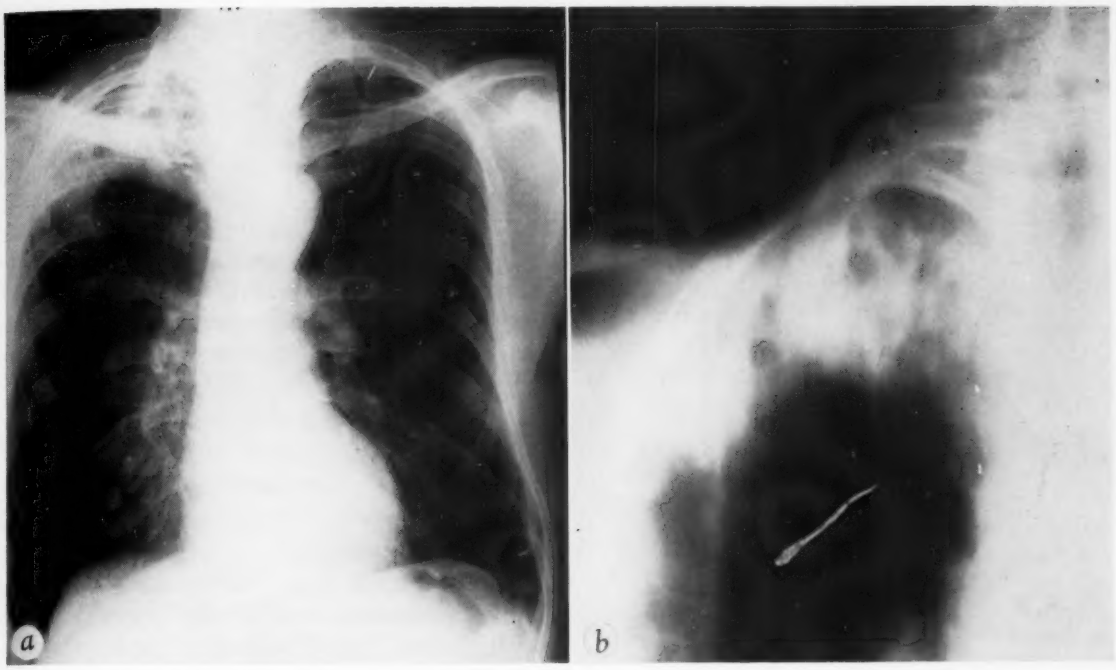

Fig. 5. Case v. (*a*) Evidence of extensive disease in upper lobe of right lung. (*b*) Laminagram demonstrating several cavities.

Five years later, in 1950, she experienced an attack of influenza, at which time her temperature rose as high as 102° F. She spent the next three months in bed. The cough persisted; the sputum became thick and yellow and continued so to the time of her examination.

Again in 1950, after a period of weakness, she consulted another physician who made a roentgenogram of the thorax and told her that she had a small "spot" in the right lung. Smears of her sputum were reported to contain acid-fast bacilli. She spent the next year in a sanatorium being treated for tuberculosis by rest in bed and dihydrostreptomycin. Twice each week since that time she had been taking a drug containing some form of streptomycin.

Smears of her sputum had been examined at three-month intervals since her dismissal from the sanatorium, and several specimens had been reported to contain acid-fast bacilli. At no time, however, did these organisms cause the death of a guinea pig and at no time did a tuberculin test give a positive result.

While in the sanatorium a bronchogram was made which was reported to demonstrate bronchiectasis in the left lung. Because of this, a program of postural drainage was prescribed.

Examination at the clinic disclosed a well-nourished woman weighing 105 pounds. The lungs were clear to percussion and auscultation. Roentgenograms of the thorax, when compared with those made elsewhere in 1950 and 1951, demonstrated a pathologic process in the right lung which had improved during the five year interval. A tuberculin test gave negative results. A smear of the sputum was reported to show acid-fast bacilli and cultures of the same material were positive for acid-fast bacilli which were not *Mycobacterium tuberculosis*. Sputum also was inoculated into guinea pigs which subsequently did not die of disease. When they were killed and examined, evidence of tuberculosis could not be found.

The patient went home and returned for examination August 9, 1956, at which time the same organism was present in the sputum. She felt reasonably well at this time.

Comment. This is another instance in which numerous cultures and virulence tests failed to provide evidence of tuberculosis. The patient received prolonged treatment with dihydrostreptomycin and, while there was roentgenologic evidence of improvement, the nontuberculous acid-fast organisms continued to be present in the sputum. The negative result of the tuberculin test should have suggested that this patient did not have tuberculosis.

FIG. 6. Case VII. Extensive disease in both lungs with cavities in both apices.

CASE VII. A white man, forty-nine years of age, was first examined at the Mayo Clinic on June 19, 1957. He had been treated for pulmonary disease since December, 1954, but had first learned of his condition in 1950 after a roentgenogram made by a mobile unit was said to show evidence of tuberculosis.

His symptoms began with an attack of so-called pleurisy in December, 1954. A roentgenogram of the thorax revealed evidence of spontaneous pneumothorax on the right; subsequent examinations disclosed that the right lung had re-expanded, although the patient had not had specific treatment for the condition. Prior to May, 1957, the patient had spent at least three periods of several months each in a sanatorium. Treatment included courses of various antimicrobial drugs and collapse of the right lung for a two month period. Many specimens of sputum were examined during this interval and each one was found to contain acid-fast bacilli. Cultures resulted in the growth of a photochromogenic mycobacterium which was not virulent for guinea pigs. At no time did a tuberculin skin test give a positive result.

Examination at the clinic confirmed the diagnosis made at the sanatorium. A roentgenogram of the thorax (Fig. 6) was interpreted as showing evidence of far advanced cavitating tuberculosis. Acid-fast bacilli were found in several specimens of sputum. A photochromogenic mycobacterium was cultured from the sputum and this microorganism, when inoculated into guinea pigs, did not produce tuberculosis. The patient was informed that his pulmonary disease was not tuberculosis.

Since his examination the patient has entered another sanatorium. A letter from physicians at the institution stated that they too had found the same organism, which they reported to be resistant to streptomycin, para-aminosalicylic acid and isoniazid.

Comment. In this case pulmonary disease has progressed in spite of intensive treatment with a variety of antimicrobial agents. At no time has the sputum failed to show the same photochromogenic acid-fast organism. That the patient's disease is not pulmonary tuberculosis seems fairly well proved.

GENERAL COMMENT

Although cases of this nature are not so common as cases of pulmonary tuberculosis, they are probably commoner than is generally realized. Timpe and Runyon[7] have reported a series of 88 cases; Crow and co-workers[2] have studied 69 patients in Georgia, and Hardy and co-workers[4] have recently listed 108 cases discovered in Florida.

So far no good designation for the microorganisms common to this condition has been suggested. Hardy and associates[4] reluctantly called them "atypical acid-fast bacilli." Weed, McDonald and Needham[6] called them "saprophytes" since, they argued, the bacteria may be quite typical for their particular strain and not "atypical" at all. However, there is reason to believe, as demonstrated by some of our cases, that the organisms cause pulmonary disease in human beings. Therefore, it does not seem exactly correct to call them saprophytes. Runyon[5] has suggested that they be designated "anonymous mycobacteria," since most strains have not yet been accurately identified or given a specific name.

Hardy and associates[4] offered the follow-

ing classification: (1) *photochromogens*, which produce buff or cream-colored colonies when grown in darkness, but which develop lemon-yellow colonies when allowed to grow in the light; (2) *nonphotochromogens*, which produce buff or cream-colored colonies wherever they are grown; and (3) *scotochromogens*, which produce yellow or orange colonies wherever they are grown.

The photochromogens and the nonphotochromogens are most commonly associated with pulmonary disease. The scotochromogens are commonly encountered but rarely in association with pulmonary lesions.

While all three types of microorganisms do not produce generalized disease in guinea pigs, photochromogens and some nonphotochromogens are pathogenic for mice. Most of the strains in all three types are resistant to isoniazid and para-aminosalicylic acid, though they are only partially resistant to streptomycin. This is in contrast to the susceptibility of *Mycobacterium tuberculosis* to these drugs. For further information on the bacteriologic characteristics of these microorganisms, the reader is referred to the articles by Hardy and associates,[4] Crow and associates,[2] Timpe and Runyon,[7] Tarshis and Frisch,[6] and Wolinsky and associates.[9]

Investigators who are familiar with the subject have emphasized repeatedly that the clinical features of the pulmonary disease associated with these bacilli cannot be distinguished from those of tuberculosis. This seems to be especially true of the roentgenographic appearance. Our limited experience supports this opinion.

CONCLUSION

The purpose of this communication, therefore, is to stress to roentgenologists and to others, who may not be familiar with the bacteriologic details, that the presence of acid-fast bacilli in a stained smear of sputum does not confirm a clinical or roentgenologic impression of pulmonary tuberculosis, but that these same acid-fast bacilli must be studied bacteriologically to determine their cultural characteristics and virulence for laboratory animals. Only after this is done can a correct diagnosis be made and proper treatment planned.

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THE CHANGING ROENTGENOLOGIC PICTURE IN PULMONARY TUBERCULOSIS UNDER MODERN CHEMOTHERAPY

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THE advent of specific antituberculosis chemotherapy has profoundly altered the course and prognosis in pulmonary tuberculosis. It is the purpose of this paper to emphasize the rapid, dramatic roentgenologic improvement that may occur under the influence of a specific therapeutic regimen in the various forms of acute pulmonary tuberculosis. Although this is an accepted fact in institutions caring for large numbers of tuberculosis patients, it is not as well known by the radiologists in general hospitals.

Prior to chemotherapy, the mortality of patients with tuberculous cavities or acute miliary disease was greater than 50 per cent in two years and approximated 100 per cent in five to ten years.[7,15,27,38] Cavity closure in large series varied from 5 per cent to 10 per cent.[5,23] Nearly 50 per cent of arrested cases were destined to suffer relapse. With present techniques, survival rates approximate 90 per cent in acute disease.[29]

Cases have been selected to illustrate the rapid, marked roentgenologic improvement which may occur in a short period of time after chemotherapy. The types of tuberculosis have been grouped according to the predominant roentgen characteristic as follows:

1. *Exudative—Productive.* Varies from the feathery, irregular, dense somewhat hazily outlined lesions of exudative disease to the dense, sharply defined, discrete shadows more typical of productive lesions.

2. *Cavitation.* Often rounded, ragged, with complete loss of lung margins and areas of increased density around its margin.

3. *Miliary—Hematogenous.* Numerous small nodules scattered diffusely throughout both lungs.

4. *Effusion.* The predominant feature being a serous effusion, usually pleural, occasionally pericardial.

5. *Pneumonia.* Resembling lobar or bronchial but more widely distributed and less well demarcated as to lobes and lobules.

The chronic caseous fibrotic and fibroid types which represent lesions of long duration have been omitted.

Numerous authorities have shown that the more recent the onset of disease and the sooner the start of chemotherapy, the more consistent and complete is the resolution of the exudative lesion. A number of investigators[1,18,30] have demonstrated that purely exudative lesions, when treated early on satisfactory regimens, may resolve completely. Small residual infiltrations usually represent an older focus which may have been present prior to the exudative process. When fibrocaseous and fibroproductive tuberculosis was present with the exudative process, streptomycin usually caused resolution of the exudative lesion and limited the fibrocaseous and fibroproductive lesions to the parenchyma already involved. Salkin and Schwartz[34] treated 106 patients with isoniazid (INH) for three to fifteen months utilizing a similar classification. Their results were as follows: exudative lesions, 64 per cent improved; exudative productive, 37 per cent improved; productive, 14 per cent improved; productive fibrotic, 4 per cent improved.

This again emphasizes that the greatest improvement occurs in the fresh, localized, exudative infiltration, while the poorest re-

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sults are obtained in the fibroproductive and fibrocavernous lesions.

1. EXUDATIVE PRODUCTIVE LESIONS

The following cases are illustrative of the exudative productive type of lesion and the response which was seen with prompt and thorough chemotherapy.

REPORT OF CASES

Case 1. J.M., a thirty-two year old white male, was admitted with a six week history of fever, productive cough, chills, night sweats, hemoptysis and a ten pound weight loss. A roentgenogram of the chest (Fig. 1*A*) revealed extensive involvement of the entire right lung, more pronounced in the right upper lobe. The lesions were primarily of varying size and in some areas were confluent. There were multiple small cavities in the right upper lobe. Multiple positive sputa were obtained and the patient was placed on INH and PAS. A roentgenogram (Fig. 1*B*) taken one year after commencement of therapy demonstrated an essentially normal chest. Very minute fibrotic residuals were present in the upper lobe.

Case 11. J.B. was admitted with a twelve day history of chills, fever, productive cough and right anterior pleuritic pain. There was a history of a ten pound weight loss in one month. Several positive sputa were obtained on admission. A roentgenogram of the chest (Fig. 2*A*) revealed an extensive infiltration of the right upper lobe with elevation of the lesser fissure. The lesion was primarily productive in nature, with several exudative areas just above the fissure. A minimal element of atelectasis was present. A roentgenogram (Fig. 2*B*) taken six months after commencement of therapy with INH, streptomycin and PAS disclosed only a

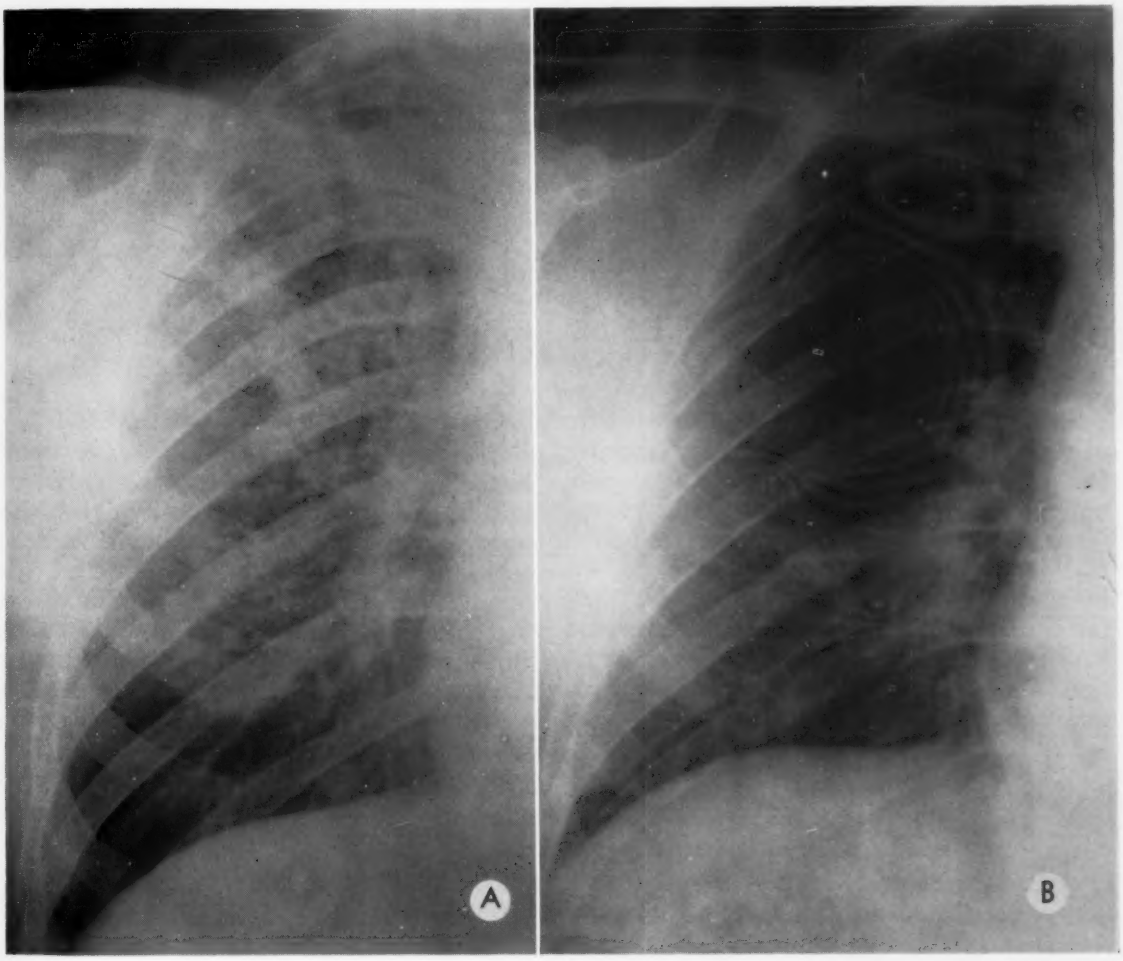

Fig. 1. Case 1. (*A*) Roentgenogram showing the extensive involvement of entire right lung. (*B*) One year later, roentgenogram shows normal chest.

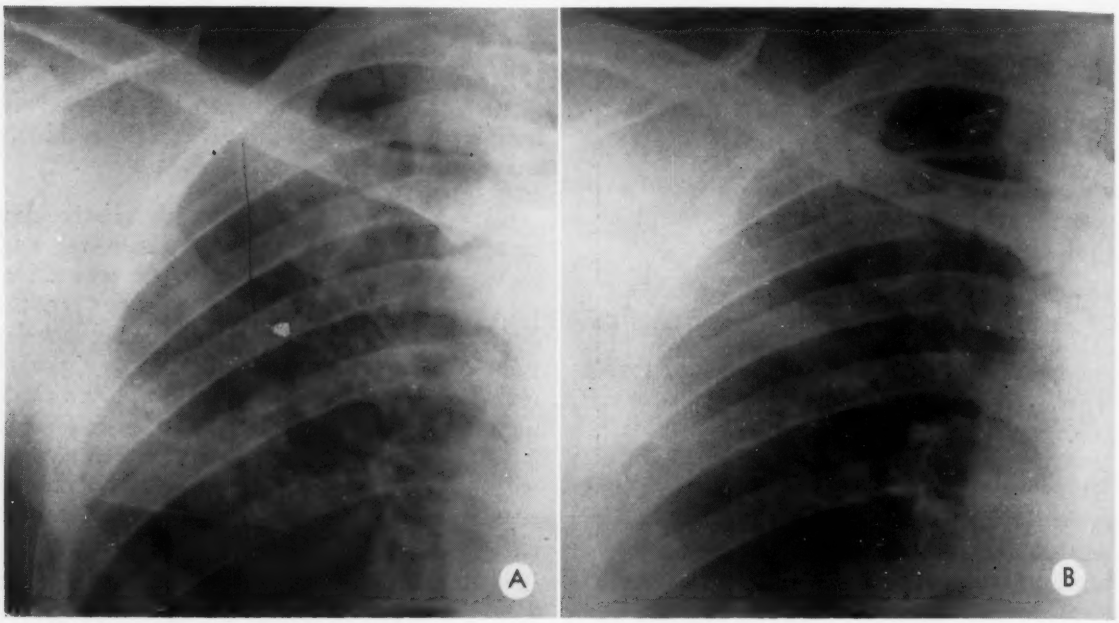

FIG. 2. Case II. (*A*) Roentgenogram showing extensive infiltration of right upper lobe with elevation o lesser fissure. (*B*) Six months later, roentgenogram shows only minimal fibrotic residual.

minimal fibrotic residual. The lesser fissure remained slightly elevated and fixed in position. There was no evidence of active disease.

CASE III. R.R., a twenty-eight year old Puerto Rican female, presented an eleven day history of cough, chest pain, hemoptysis, night sweats, fever and anorexia. Past history revealed two previous episodes of hemoptysis several years prior to the present admission. The Mantoux test was positive at 1:1000. No positive sputa were obtained. A roentgenogram of the chest (Fig. 3*A*) revealed a very extensive lesion consisting of productive nodules throughout the entire right lung. Except for pleural diaphragmatic adhesions at the base and minimal fibrotic infiltrations at the apex, the left lung was clear. A roentgenogram (Fig. 3*B*) taken after five months of streptomycin and PAS therapy showed clearing of the entire lesion. There was no residual to suggest the extensive nature of the original lesion.

2. CAVITATION

Many reports have been published on the efficacy of antituberculous therapy in effecting the rapid closing of cavities.[26,31,33] The over-all figures indicate that up to 85 per cent of single cavities will disappear, the majority within eight months after the initiation of therapy. Of those patients with two or more cavities, 55 per cent will disappear under a good regimen. The usual therapy for cavitary disease includes INH alone, INH with streptomycin or INH and PAS. Several reports[13,24,31] show that up to 40 per cent may close within three months of onset.

REPORT OF CASES

CASE IV. L.P., a thirty-four year old Puerto Rican male, was admitted with a two month history of productive cough and minimal weight loss. The Mantoux test was positive but sputa were negative. On the initial examination, a well defined cavity was present in the right upper lobe measuring about 4 cm. in diameter with a thick 4 mm. wall (Fig. 4*A*). A minimal amount of interstitial reaction was evident, extending from the cavity to the hilar area. It was felt that this represented a solitary cavitary lesion. The patient was placed on triple therapy with streptomycin, PAS and INH. Immediately after the commencement of therapy, three positive sputa were obtained. Six months later (Fig. 4*B*), the cavity was healed. A fibrotic residual lesion was present in the right upper lobe.

A further variant in cavitary healing, commonly called open cavitary healing, has

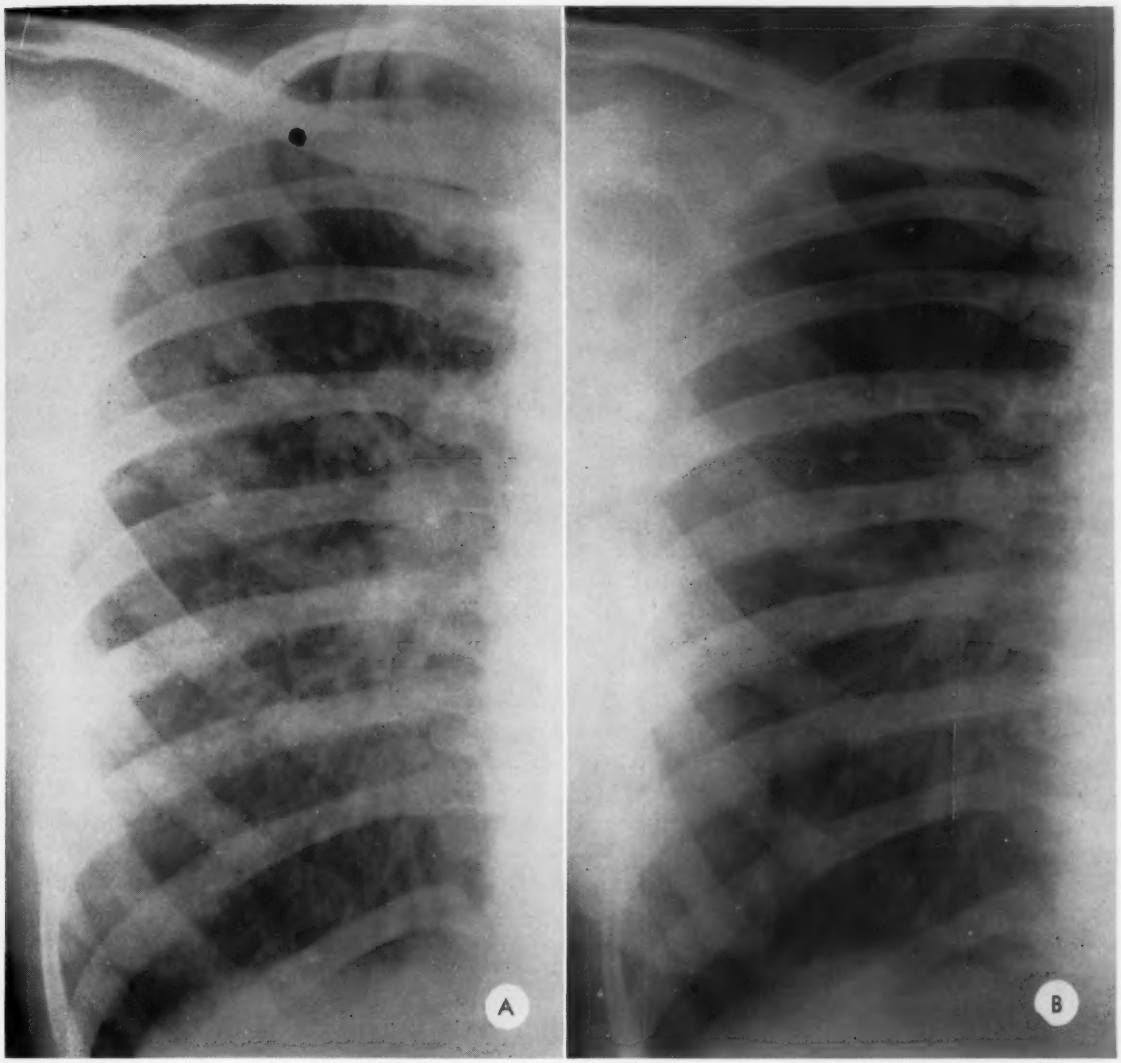

Fig. 3. Case III. (*A*) Roentgenogram showing the extensive lesion consisting of productive nodules throughout entire right lung. (*B*) Five months later, roentgenogram shows clearing of entire lesion.

become a more prominent feature since the advent of INH therapy. Instead of resolution of the cavity as in Case IV, a persistent sterile thin walled cavity remains. Thompson,[37] in reviewing 240 surgically resected specimens containing cavitary lesions, classified 23 (9.6 per cent) as exhibiting open cavitary healing. Of these 23 cases, 20 had received INH, the remaining 3 streptomycin and PAS. Most authors[2,4,11,19,35] agree that open cavitary healing seems to be a phenomenon primarily associated with INH therapy. Auerbach and Small,[4] in more than 2,000 autopsies prior to the advent of antituberculous chemotherapy, noted only 2 cases of open cavitary healing, while 12 such cases occurred in 98 cavities resected following chemotherapy.

Case V. K.K., a seventeen year old white male, was admitted with a two year history of fever, night sweats, anorexia, right chest pain and weight loss. Multiple sputa were positive for tuberculosis. The initial roentgenograms (Fig. 5, *A* and *B*) showed an extensive consolidation of the right upper lobe with elevation of the lesser fissure. The lateral roentgenogram demonstrated a large thick walled cavity in the apical segment. The patient was placed on

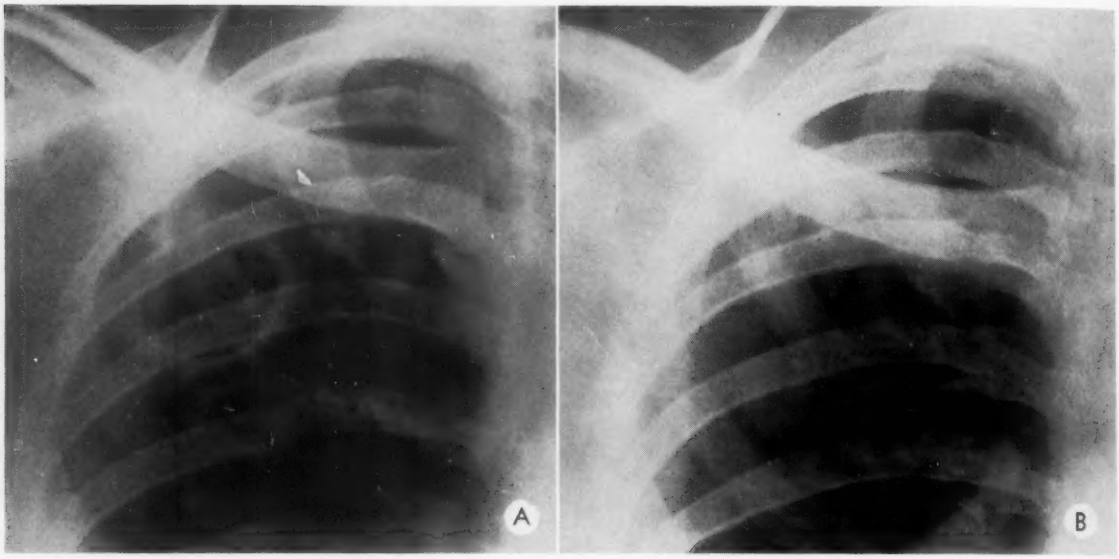

FIG. 4. Case IV. (*A*) Roentgenogram showing a well defined cavity in right upper lobe measuring about 4 cm. in diameter. (*B*) Six months later, roentgenogram shows healing of cavity.

triple therapy. A roentgenogram of the chest later (Fig. 5*C*) revealed complete resolution of the consolidation. A large thin walled cavity typical of the type seen in open cavitary healing occupied the site of the original thick walled cavity (Fig. 5*D*). Sputa were persistently negative.

3. MILIARY LESIONS

In acute disseminated miliary disease treated with streptomycin alone, Bunn[6] reported 15 patients surviving out of a total of 22. In 9 of the 15 (41 per cent), complete resolution of the pulmonary lesion was noted. In 8 cases of miliary tuberculosis treated with 3 to 6 gm. of streptomycin per day, McDermott and co-workers[25] reported complete clearing in 6 cases after five months of therapy. Several other authors[8,12] have published series of cases which have shown complete disappearance of lesions in time ranging from two to five months. As previously mentioned, the Committee of Chemotherapy and Antibiotics[29] has reported survival rates of 90 per cent and above with the use of INH as part of the drug regimen.

REPORT OF A CASE

CASE VI. W.W., a forty-nine year old Negro male, was admitted with a one year history of anorexia, weakness, cough and a thirty pound weight loss. Multiple positive sputa were obtained at the time of admission. A roentgenogram (Fig. 6*A*) revealed a very extensive productive lesion involving the entire right and left lung, with a minimal interstitial reaction. It was felt that the patient had hematogenous miliary tuberculosis, associated with an interstititial reaction. During the initial work-up, the patient complained of dysphagia, and a carcinoma of the middle third of the esophagus was discovered. The patient was placed on streptomycin and INH. Six months after commencing therapy, there was complete resolution of the entire lesion with only a minimal amount of fibrotic residual confined primarily to the left upper lobe (Fig. 6*B*). A diaphragmatic adhesion at the left base was present on the initial roentgenogram. Shortly after this examination, the patient expired from the carcinoma. Postmortem examination revealed a moderate, diffuse, bilateral interstititial fibrosis in addition to the carcinoma of the esophagus but no evidence of active tuberculous tissue.

4. PLEURAL EFFUSION

Roper and Waring[32] in 141 cases of serofibrinous tuberculous pleural effusion treated with bed rest and no chemotherapy reported that the usual time required for complete reabsorption ranged from approximately two months for the small effusion to four months for the larger ones. Hinshaw and Garland[16] stated that there is a con-

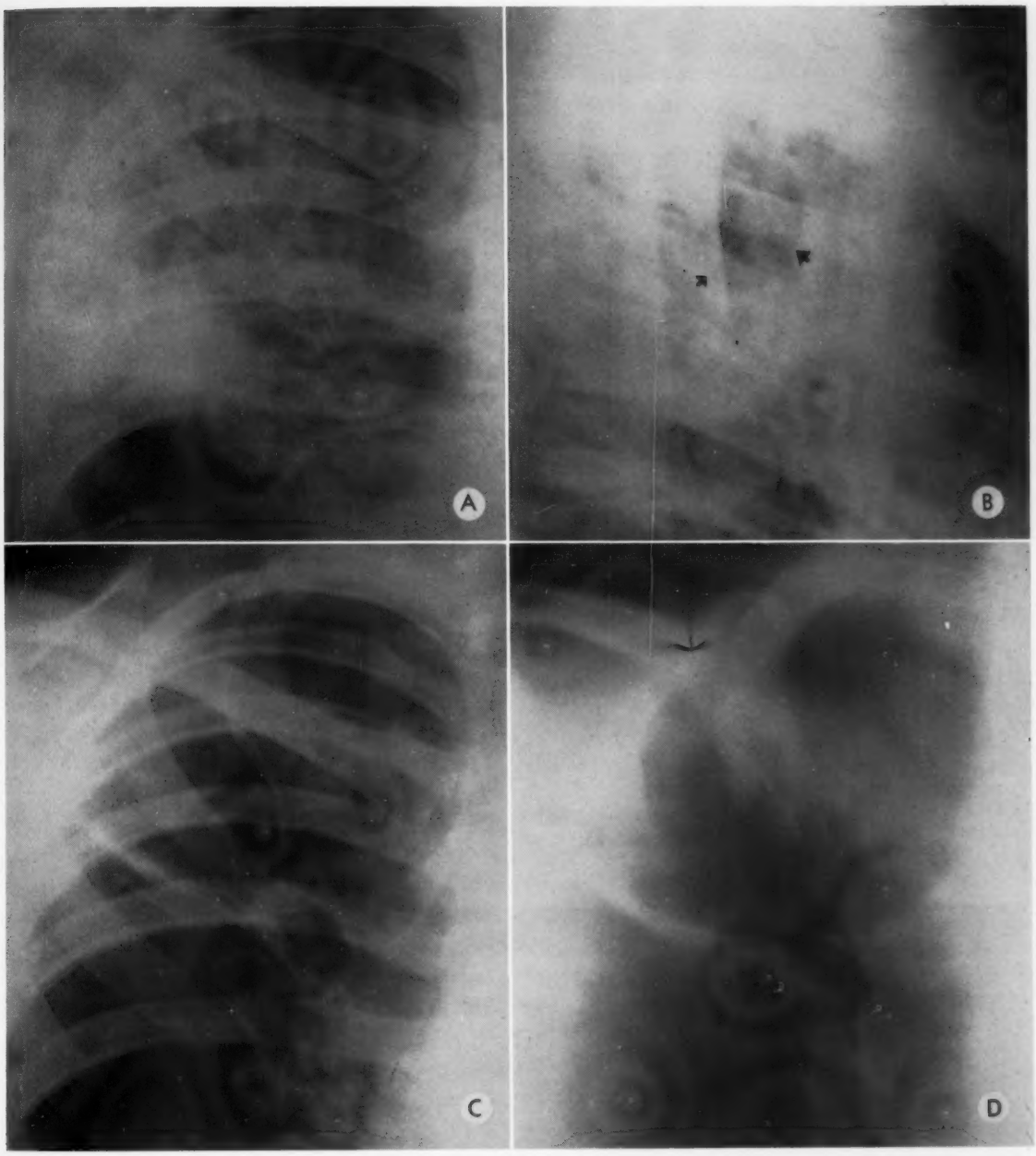

Fig. 5. Case v. (*A* and *B*) Roentgenograms showing extensive consolidation of right upper lobe with elevation of lesser fissure. (*B*) Lateral roentgenogram shows a large thick walled cavity in apical segment. (*C*) Roentgenogram showing resolution of the disease process after therapy. (*D*) Roentgenogram showing thin walled cavity occupying site of original thick walled cavity.

siderable variation in the rate of absorption of pleural fluid which seems to be related to the duration of the effusion. With chemotherapy, however, the fluid steadily diminishes and disappears within six to eight weeks. LeBecq and Tirzmalis[22] treated serofibrinous pleural tuberculous effusions with ACTH, INH and PAS. They reported spectacular, rapid results with the effusion being absorbed in an average period of twelve days following initiation of therapy. Even *et al.*[14] treated 160 cases of tuber-

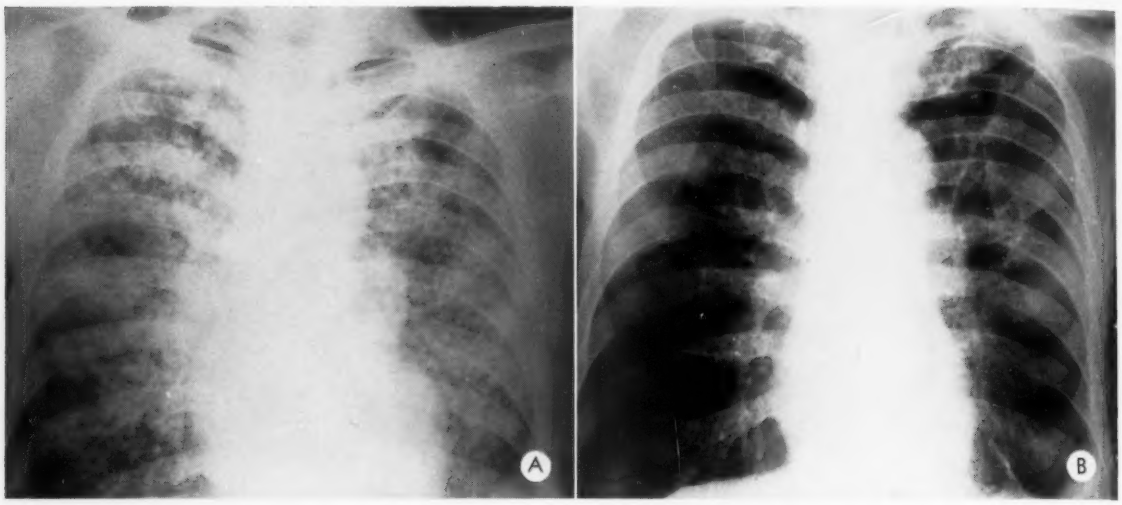

FIG. 6. Case VI. (*A*) Roentgenogram showing extensive productive lesion involving entire right and left lung. (*B*) Six months later, roentgenogram shows complete resolution of entire lesion with only a minimal amount of fibrotic residual.

culosis with ACTH, cortisone, INH and streptomycin and recorded their most satisfactory results in 54 cases of acute tuberculous serous effusions (mostly pleural) with cure being obtained rapidly.

REPORT OF A CASE

CASE VII. M.R., a thirty-two year old Negro female, was transferred from another institution with a history of productive cough, fever, dyspnea, and hemoptysis. Roentgen examination revealed a pleural and pericardial effusion and a pericardial tap disclosed tubercle bacilli. Triple therapy was initiated prior to transfer. On admission to the hospital, a roentgenogram (Fig. 7*A*) demonstrated two areas of loculated pleural effusion, one on the right extending up along the lateral costal margin into the lesser fissure and one on the left probably representing an infrapulmonary effusion between the lung and diaphragm. In addition, pericardial

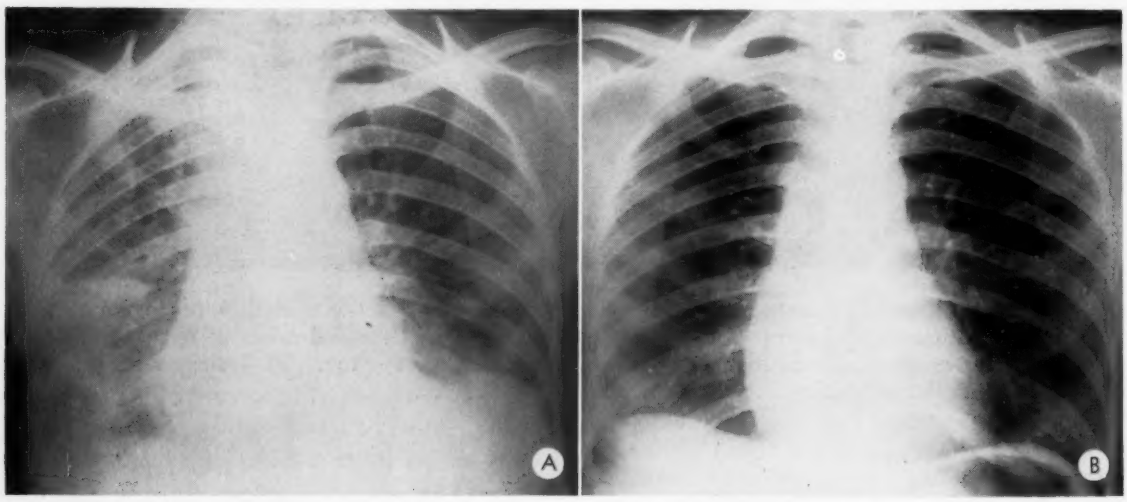

FIG. 7. Case VII. (*A*) Roentgenogram showing two areas of loculated pleural effusion, one on the right extending up along lateral costal margin into lesser fissure and one on the left, probably representing an infrapulmonary effusion between lung and diaphragm. (*B*) One month later, roentgenogram shows reabsorption of fluid from pericardial and pleural spaces with only mininal pleural thickening along right costal margin.

effusion had been proved at the previous institution. Physical examination revealed a pulse rate of 118, respiration of 20, an elevated temperature, and a blood pressure of 122/84. The electrocardiogram was compatible with pericarditis. Venous pressure was 190 mm. of saline. The sedimentation rate was 41; the white blood cell count was 18,300, with a normal differential. A pleural tap was performed and culture revealed acid fast bacilli. Triple therapy was continued with the addition of metacortin. Venous pressure dropped to 90 and the electrocardiogram became normal. The sedimentation rate was 14 at the time of discharge. A roentgenogram (Fig. 7*B*) taken only one month after admission, demonstrated dramatic reabsorption of fluid from the pericardial and pleural spaces with only minimal pleural thickening along the right costal margin.

5. TUBERCULOUS PNEUMONIA

Lobar consolidation in the acute stage will often respond dramatically to conventional therapy.

REPORT OF CASES

Case VIII. W.L., a thirty-three year old Negro male, was admitted to another institution with a two month history of productive cough and weakness. A roentgenogram (Fig. 8*A*) demonstrated an extensive pneumonic process involving the entire right upper lobe which was evidently associated with a minimal component of atelectasis as the lesser fissure was elevated superiorly. A minimal infiltration was present in the right lower lobe as well. The left lung showed no evidence of active disease. Sputum was positive and the patient was placed on INH and streptomycin. A roentgenogram taken four months after admission (Fig. 8*B*) revealed almost complete clearing of the right upper lobe with a minimal fibrotic residual. The right hilar region had retracted somewhat superiorly. The lesser fissure remained slightly elevated.

Recently many authors have reported improved results with the addition of steroids to the standard antituberculous chemotherapy. Cochran *et al.*[10] treated 19 patients with antituberculous chemotherapy and cortisone. Eleven of the 19 were considered to have shown accelerated improvement with the best results occurring in the relatively acute lesions. Climie *et al.*[9] added corticotrophin to the therapy of 3 patients previously deteriorating on streptomycin, PAS, or INH and reported marked clinical and roentgenologic improvement. Even *et al.*[14] treated 25 patients with parenchymal infiltrations with antituberculous therapy, cortisone and ACTH and reported 12 cured and 11 improved. Similar therapy has been applied to nodular and cavitary lesions with apparent ac-

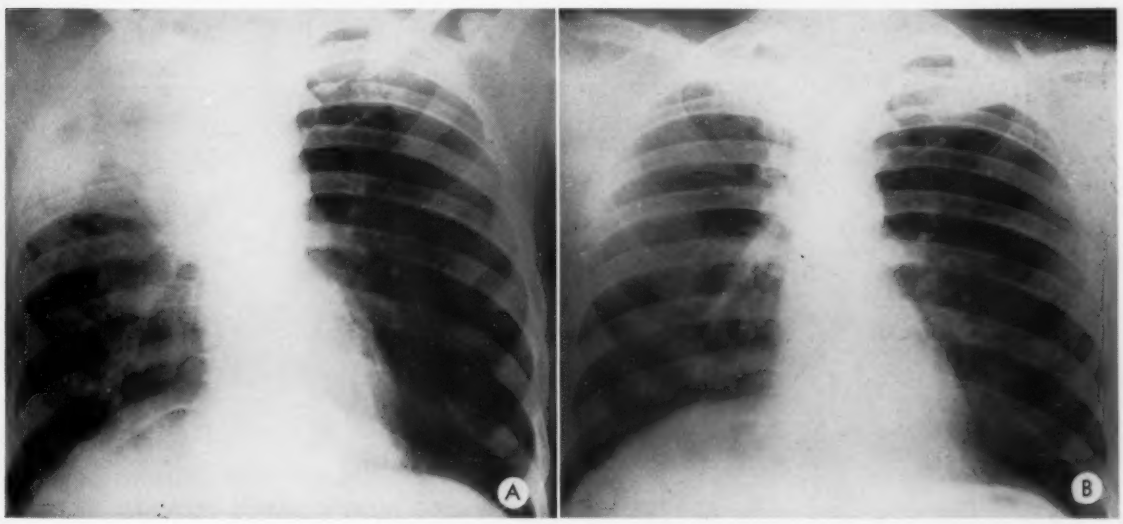

Fig. 8. Case VIII. (*A*) Roentgenogram showing extensive pneumonic process involving entire right upper lobe. (*B*) Four months later, roentgenogram shows almost complete clearing of right upper lobe.

celerated improvement being noted clinically and roentgenologically.

CASE IX. R.G., a thirty year old white male, a chronic alcoholic, was admitted with a history of chills, night sweats, weight loss, cough and hemoptysis. A chest roentgenogram (Fig. 9*A*) revealed advanced pulmonary tuberculosis with cavitation. He was treated for four months with streptomycin and PAS with no significant resolution, following which he left the hospital against advice. Sputa were persistently positive. The patient was re-admitted in a moribund state, one month later. Metacortin was added to his therapeutic regimen. Within the following two months, there was a marked improvement in his general condition with a fifty pound weight gain. Sputa became negative and steroids were discontinued. A subsequent roentgenogram (Fig. 9*B*) revealed marked resolution of the exudative process in both lungs. Residual chronic fibrotic infiltrations persisted along with bronchiectatic changes. The patient remained relatively asymptomatic.

DISCUSSION

The series of cases presented illustrate the dramatic roentgenologic improvement which can occur in pulmonary tuberculosis under the influence of chemotherapy. It is the potentially reversible lesion of recent origin in which antimicrobial therapy will enhance reabsorption and prevent the multiplication of bacilli, leading to caseation, necrosis and progressively severe tissue destruction. This reversal of the progressive trend of the disease, which may be achieved early by chemotherapy, is most strikingly illustrated by the patient whose disease is characterized by an acute onset and roentgen findings of extensive pneumonic involvement, frequently with evidence of apparently fresh cavity formation.[31]

Tempel and co-workers[36] state that the most important single factor in prognosticating therapeutic response to drug therapy appears to be the duration of the disease process prior to the institution of antimicrobial therapy with the results most apt to be definitive in patients with disease of recent origin. The maximum effect of chemotherapy is obtained at the stage of exudation and local cell proliferation.[20] In the presence of caseonecrotic and fibrotic changes, it may take many months before improvement is noted and healing may be incomplete.[35] The clinical observations of the British Tuberculosis Association[17] and the Co-Operative Investigation of the Veterans Administration[39] indicate that the more advanced the disease, the less satisfactory the results. The lack of change is due to the inability of the drugs to penetrate or have an effect on tissue; thus the anatomic changes become permanent. Auerbach[3] compared the treated and untreated lesions of more than 2,400 autopsy

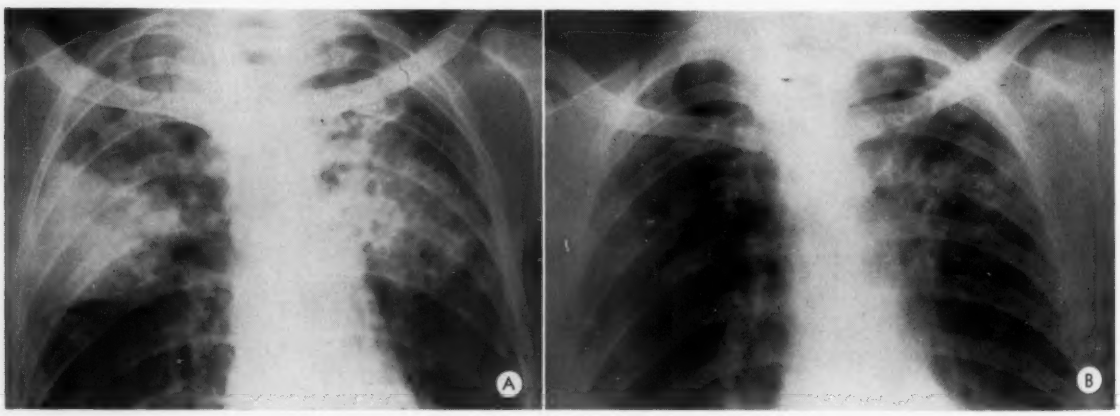

FIG. 9. Case IX. (*A*) Roentgenogram showing advanced pulmonary tuberculosis with cavitation. (*B*) Roentgenogram showing marked resolution of exudative process after therapy.

and 500 surgical specimens. He observed the following differences after chemotherapy which are secondary to the rapid clearing of the perifocal reaction by specific drugs: (a) the capsules around necrotic foci are thinner; (b) the walls of the cavities are thinner; there is (c) a decrease in pulmonary fibrosis and emphysema; and (d) open cavity healing occurs with greater frequency.

Rapid and extensive clearing of the perifocal reaction is particularly striking in the cases in which the process is acute and extensive. The rapid clearing of the perifocal reaction on serial roentgenograms can be easily observed, and marked changes can be seen in a matter of weeks. Kumar[21] reported 3 cases of bilateral far-advanced pulmonary tuberculosis in which pathologic examination of the resected tissue after chemotherapy did not reveal any evidence of tuberculosis. This clearing is of importance since, if it does not occur, the perifocal reaction will end in fibrosis or necrosis.

An anatomic study of 13 resected lungs treated with antimicrobials and steroids indicates that ACTH and cortisone cause the disappearance or notable diminution of nonspecific exudative lesions, permitting the full use of antibiotics.[14] Studies in experimental tuberculosis prove that ACTH and cortisone do not inhibit but reinforce the effect of antibiotics.

SUMMARY

Emphasis has been placed upon the rapid resolution noted roentgenologically in pulmonary tuberculosis under modern chemotherapy. Productive, exudative, miliary, pneumonic and cavitary forms of tuberculosis respond dramatically, if marked fibrosis and caseation has not occurred. Many long standing cases of chronic disease without extensive fibrosis show rapid and marked improvement under proper therapy.

The extent of involvement does not affect the prognosis of the disease today as it did before modern antimicrobial therapy. The presence, however, of abnormal anatomic changes of extensive fibrosis or caseation must still be regarded as a poor prognostic sign.

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Vol. 81, No. 2

THE GHON FOCUS OF TUBERCULOUS REINFECTION*

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THE typical roentgenographic appearance of the active primary tuberculous complex is a dumbbell shadow, one end of which is the parenchymal focus and the other the lymph node component. In due course of time, both shadows become smaller, denser, and more sharply delineated, and finally undergo calcification and ossification. The size of these round calcific deposits is usually 0.5 cm. or less in diameter. Development of a primary complex follows tuberculous infection in the vast majority of the cases as proved on autopsies; however, it often cannot be demonstrated roentgenographically because of its small size or hidden location. Not infrequently, the complexes are "incomplete," *i.e.*, either the parenchymal or the lymph node component is missing, and in other instances multiple primary complexes are found. The term "Ghon focus" is still frequently used for the parenchymal component, although Ghon did not claim to be the first one to describe these lesions. Calcification, as a rule, develops in one or more years, but in infants who were accidentally inoculated with virulent organisms in Luebeck, Germany, incipient calcification was noted as early as two months after the inoculation, and complete calcification after eight months in some of the cases. Development of calcification is much slower in adults; it usually takes four or more years.

Anatomic study on innumerable cases seems to establish the fact that a primary complex is structurally characteristic and pathognomonic for the primary infection. Until 1940, it was generally accepted that any subsequent infection with tubercle bacilli would result in the reinfection-type of reaction, and a second primary complex would never be formed. In that year, Terplan[11] reported on 29 autopsies in which formation of typical complexes due to reinfection was found after complete healing of the real primary complex. Apparently, in these cases, the body, in spite of a prior infection, returned to the same type of reactivity that existed before the first infection had taken place. In a subsequent article in 1946,[12] he described another 29 cases which fell into this category and drew the conclusion that a pulmonary reinfection complex should not be considered as an occurrence of great rarity, but added that considerably larger material was needed in order to gain more accurate information. Rich[9] believes that there is room for a difference of opinion regarding the conclusion that all of the cases described by Terplan were instances of reinfection. He is of the opinion that while occasional individuals may lose their resistance conferred by primary infection to the extent that they will respond to reinfection with the development of a second primary-type pulmonary lesion, the result of studies all over the world has shown this event to be decidedly uncommon, regardless of the degree of exposure to infection. Once a primary infection has been established, the body rarely returns to its originally native state of resistance. Schwartz,[10] in his autopsy material, found instances of primary complexes at different ages in the same individual. He recommended the use of the term "initial complex" versus "primary complex." He expressed the view that the entire cycle of infection, allergy, healing, and development of immunity takes three to five years. After this time the organism might become ready for another infection which again would take the characteristic shape of the primary complex. Pagel and Toussaint[7] pointed out that the problem of the reinfection complex is clear cut when an entirely healed primary complex is found in the lungs and a recent tuberculous lesion in a different organ, but it becomes

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more difficult when these two sets of lesions occur in the lungs. In the diagnosis of reinfection, error may arise from the following two sources: (1) endogenous origin of recent looking changes from an obsolete lesion, (2) delayed calcification of both parts or asynchronous calcification of the two constituents of the primary complex. In recent years, 2 more cases[8] of reinfection complex, encountered on autopsy, were reported. Medlar[5] found reinfection complexes in 2.5 per cent of 889 autopsy cases of minimal disease and stated that they occur infrequently, but do exist, and should not be ignored.

Obviously while these lessions can be found much more frequently at autopsies than on roentgenographic examinations, the postmortem examination does not permit in every case the determination of the etiology of completely calcified lesions. Coexistence of calcifications due to histoplasmosis and tuberculosis is not rare, and in such instances the diagnosis of a Ghon focus of reinfection might be in error. On roentgenograms, calcified emboli, intraalveolar osteomas, calcified foreign bodies, etc., may resemble tuberculous complexes.

Clinical observations on Ghon foci of reinfection are not too numerous. Bloch,[2] upon discovery of small caseous foci at contact examination of asymptomatic adults, and following them for several years until residual calcification was noted, recommended re-evaluation of the problem of Ghon's tubercle, and came to the conclusion that a calcified tuberculous lesion represented an innocuous residual of a tuberculous infection, either primary or reinfection. A few cases[6,13] were reported on development of reinfection complexes in different organs (skin, conjunctiva) with roentgenographic evidence of an old calcified primary complex in the lungs. It should be noted that the majority of the pertinent observations were described at a time when it was assumed that all small round calcific foci were tuberculous in origin, and the presence of a calcified nodule with a negative tuberculin reaction presented a case of waning tuberculin sensitivity in a previously tuberculin-positive individual. However, subsequently two authentic observations[3,4] were made on development of a primary complex with positive tuberculin test, followed by waning of the tuberculin sensitivity and its return upon formation of a reinfection complex. In Badger and Ayvazian's paper,[1] the case of a tuberculin positive Negro student nurse is described, who developed a small parenchymal focus which calcified subsequently, thereby forming a Ghon focus of reinfection. In the following case, both tuberculin and fungus skin tests were done prior to and after the development of reinfection complex.

REPORT OF A CASE OF "PSEUDO-PRIMARY" COMPLEX

A fifty-seven year old white man had his first tuberculin test (cutaneous, Pirquet) at the age of twenty-five. It was positive (2 plus). He had never had clinical tuberculosis, but there was a history of exposure to an open case of pulmonary tuberculosis for several months in 1918. As this man, a physician, was interested in the study of anticutin tests in which he used his own serum, he tested his tuberculin sensitivity on several occasions between 1929 and 1936, and again between 1942 and 1944. The Man-

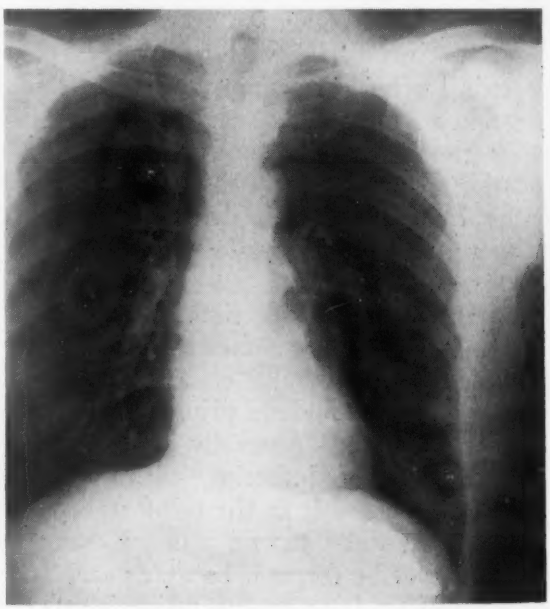

FIG. 1. Roentgenogram taken April 17, 1944 showing a calcified lymph node near the left hilus.

toux test was always positive (old tuberculin 1-10,000). An Army induction roentgenogram on April 17, 1944 revealed a calcified lymph node near the left hilus (Fig. 1). In July, 1945, while serving overseas, another routine chest roentgenogram was made following exposure to an active case. A two months' period of cough, fatigability and loss of weight, which were attributed to a cold, preceded this roentgenographic examination. It showed a small amount of soft confluent infiltration at the second anterior intercostal space on the left. He was hospitalized for two weeks during which, among several negative smears, one was noted to show two "atypical acid-fast rods." As there was significant diminution in size—but no complete resolution—of the lesion in this short period of time, he was discharged from the hospital with the diagnosis of healed bronchopneumonia. Subsequent routine semi-annual roentgenographic examinations (Fig. 3 and 4) revealed gradually increasing fibrosis and, finally, calcification of the residual lesion. Since 1954, the roentgenograms have presented the typical appearance of a calcified bipolar primary complex. Coccidioidin and histoplasmin skin tests were negative, and he never stayed in geographic areas in which these fungus diseases are endemic.

This man, who has been tuberculin positive on repeated tests for thirty-two years, had on his chest roentgenogram a long-standing calcified hilar nodule, representing the hilar component of an obsolete primary tuberculous in-

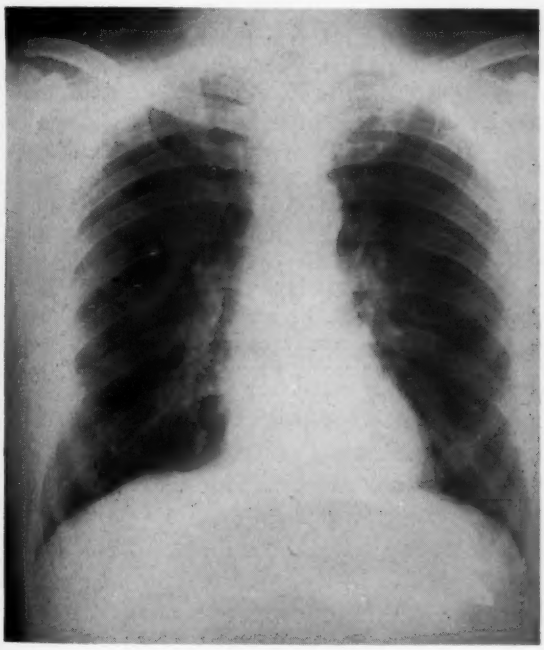

Fig. 2. November 14, 1947.

Fig. 3. August 22, 1952.

Fig. 4. Roentgenogram taken April 22, 1955 showing the typical appearance of a calcified bipolar complex.

fection with no discernible parenchymal component. Following exposure to a far-advanced active case, he developed a small parenchymal focus, which slowly and gradually calcified, and represented the parenchymal component of a "second primary" or reinfection complex with no discernible hilar component. Because, fortuitously, the obsolete hilar calcified nodule happened to lie in an area which on the roentgenogram appeared to be in the line of lymphatic drainage from the recently formed parenchymal focus, these two asynchronously formed lesions presented a roentgenographic appearance which was undistinguishable from a commonly seen calcified bipolar primary complex. A roentgenogram taken on April 22, 1955 (Fig. 4) was shown to a number of phthisiologists and radiologists without presentation of the previous roentgenograms, and it was read without exception as "negative chest, except for a calcified primary complex."

This is then a case where development of a bipolar complex was observed, the hilar component of which represented the calcified residual of an obsolete primary infection and the parenchymal one a slowly and gradually calcifying focus of reinfection.

SUMMARY

After a review of the literature on the Ghon focus of tuberculous reinfection, the case of a tuberculin-positive and histoplasmin and coccidioidin-negative individual is presented in whom the hilar component of the first infection and the parenchymal component of the reinfection assumed the reoentgenographic appearance of a bipolar primary complex. Waning of a previously present tuberculin sensitivity is not a necessary prerequisite to formation of a Ghon focus of reinfection.

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LESS FAMILIAR ROENTGEN PATTERNS OF PULMONARY GRANULOMAS*

SARCOIDOSIS, HISTOPLASMOSIS AND NONINFECTIOUS NECROTIZING GRANULOMATOSIS (WEGENER'S SYNDROME)

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SARCOIDOSIS

THE common roentgen signs of pulmonary sarcoidosis have been adequately described in the literature. There are numerous reports depicting lymph node enlargement, disseminated miliary densities, localized infiltrates, and fibrotic changes, alone or in combination. However, not all cases reveal these more or less typical findings. In this paper, attention is focused on two interesting but less familiar roentgen patterns of sarcoidosis which may, at times, bring this disease to mind.

Large Multinodular Pattern. In 1952, McCord and Hyman[35] reported 2 cases of sarcoidosis in which large pulmonary nodules were demonstrated roentgenologically. Turiaf and Brun,[50] in 1955, reproduced the roentgenograms of another case. Initially, the chest roentgenogram showed diffuse miliary lesions. Three years later these lesions had cleared, but there were now five large sharply-defined nodules in the lungs. These disappeared almost completely in the ensuing two months.

We have had the opportunity of reviewing the roentgenograms in 4 cases, including the 2 reported by McCord and Hyman. In each instance a roentgen diagnosis of pulmonary metastasis was made originally. However, the mild clinical symptoms and continued well-being of the patient belied this diagnosis, and ultimately microscopic evidence of sarcoid was obtained, twice in tissue from the lung and twice by means of a peripheral lymph node biopsy.

All 4 patients were young Negro males with mild systemic and respiratory symptoms. All had slight to moderate peripheral lymph node enlargement, and 2 showed elevation of the serum globulin and serum calcium. None presented clinical evidence of involvement of other organs.

In each case the chest roentgenogram demonstrated striking findings. Multiple round densities, 5 to 35 mm. in diameter, were widely distributed in the lungs. In 3 cases the nodules were numerous, hazy or fluffy in outline, and tended to be confluent (Fig. 1*A*). In the fourth case the nodules were fewer in number, sharper in outline, and discrete, as in the case of Turiaf and Brun. Two cases showed widening of the superior mediastinum. The presence of hilar enlargement could not be ascertained because of overlying pulmonary lesions.

Follow-up roentgenograms were available in 3 cases. No change was observed at five months in 1 case and slight improvement was noted at four months in another. The third case showed a marked change at nine months (Fig. 1*B*). The nodular densities were greatly reduced in size and there was now a fine diffuse infiltration throughout the lungs.

The pattern characterized by multiple large nodules cannot, of course, be considered diagnostic of sarcoidosis. It is a rare manifestation of this disease and more frequently a sign of metastatic neoplasm. Although the fluffy confluent lesions noted roentgenographically do not closely simulate the lesions of ordinary metastatic tumor, the resemblance to the "halo" metastatic nodules of chorionepithelioma and certain other neoplasms is striking.

Other conditions to be considered in dif-

* From the Department of Radiology, University of Cincinnati College of Medicine and the Cincinnati General Hospital. Presented at the Fifty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 1–4, 1957.

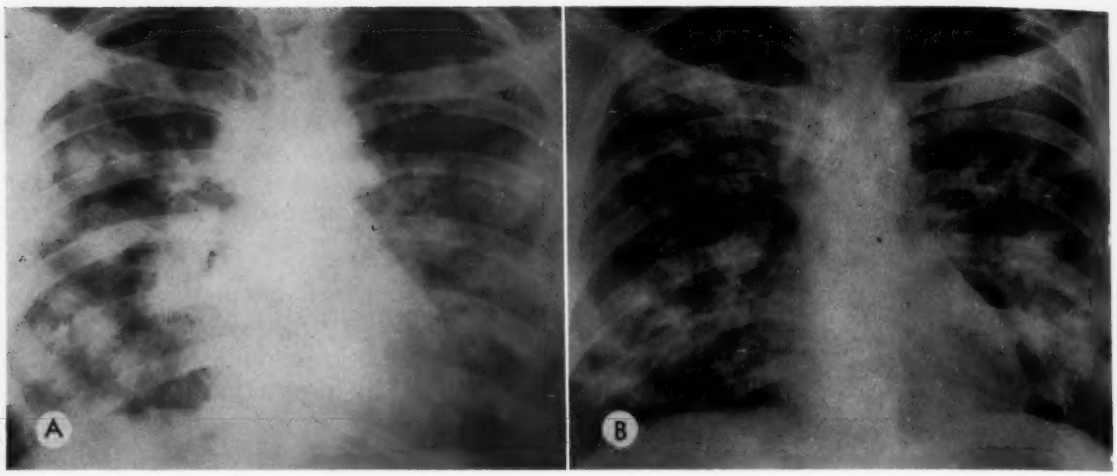

FIG. 1. Multinodular sarcoidosis. (*A*) April, 1956. The nodules are fluffy in outline and tend to be confluent. There is right superior mediastinal lymph node enlargement. (*B*) January, 1957. The nodules are greatly reduced in size and there is a fine diffuse infiltration throughout the lungs.

ferential diagnosis include multiple histoplasmomas, the rare nodular form of tuberculosis, and multiple benign tumors and hamartomas. In these conditions the nodules are generally few in number, sharp in outline, and often contain calcium.

Certain features encountered in the present cases may provide clues to the correct diagnosis. These include the apparent predilection for young male Negroes, the mild clinical symptoms, the continued state of well-being of the patient, and the presence in other body systems, especially in the lymph nodes, of stigmata consistent with sarcoidosis. Roentgenographically, the fluffy margins of the pulmonary nodules, the tendency toward confluence, and the stationary or regressive character of the lesions are of added significance.

Multicystic Pattern. This pattern of sarcoidosis is probably a complication of the irreversible fibrotic stage of the disease. Although individual cases have been reported and occasional examples have been cited in articles dealing with the roentgen manifestations of sarcoid,[2,22,36,38,43,52,54] few authors have dwelt on this phase of the disease.

Longcope and Freiman[33] stated that the large cavitary lesions seen in chest roentgenograms in sarcoidosis usually proved to be emphysematous bullae or cystic bronchiectatic dilatations. They pointed out that these changes resulted from severe pulmonary fibrosis. Turiaf and Brun[50] also related the emphysema and bronchiectasis to cicatricial fibrosis. They noted a good correlation between the extent of the emphysema and the severity of the symptoms and signs, and recorded such complications as pneumothorax and cor pulmonale. Mallory[34] reported the autopsy findings in 6 cases of long-standing pulmonary fibrosis of probable sarcoid etiology. Extensive bullous emphysema was present in all cases.

Our experience with the multicystic pattern of sarcoidosis dates back about ten years and is based on 9 proved cases, 6 from the Cincinnati General Hospital (representing 21 per cent of the 28 cases of sarcoid seen during this period), and 3 from other institutions in the Cincinnati area.[11] All the cases from the General Hospital were Negroes (about 50 per cent of the patients in this institution are Negroes) but the outside cases were white. The average age of the 9 cases was thirty-eight years. The control group of 22 cases of sarcoid in which the multicystic pattern was not manifest were all Negroes. The average age was thirty-one years.

Respiratory symptoms in the cases with multicystic disease were usually severe and

progressive. Cough and dyspnea were prominent. Only 1 case, a patient with minimal pulmonary changes, was asymptomatic. The incidence of involvement of other body structures was approximately the same in the cases with cystic lesions as in the control group.

On the initial roentgenogram multicystic lesions were the predominant feature in only 1 case, although they were present to some degree in several others. In the remaining 8 cases extensive pulmonary infiltrate was apparent, fibrotic in nature in 6 and miliary in 2. In 2 cases, 1 with fibrosis and 1 with miliary densities, the involvement appeared to be almost entirely limited to one lung. The predominantly unilateral miliary lesions exemplified an exceedingly rare distribution of this form of sarcoidosis (Fig. 2*A*). Mediastinal and/or hilar enlargement was evident on the first roentgenogram in 7 cases and appeared later in 1. Spontaneous pneumothorax occurred terminally in 1 case.

Two patients were followed for less than six months. In the other 7, chest roentgenograms were available for periods of from three to nine years. The cystic lesions, once they appeared, increased gradually in number and extent. In no instance did they re-

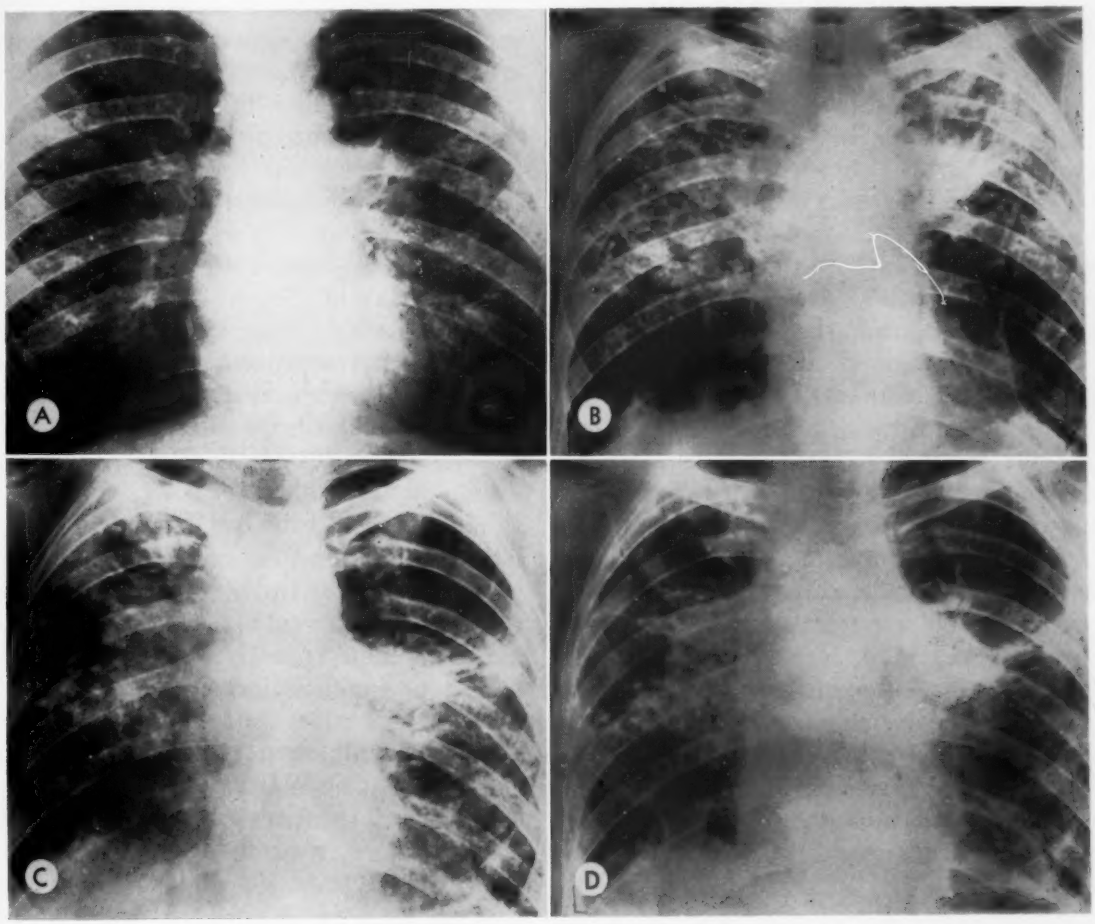

Fig. 2. Multicystic sarcoidosis. (*A*) May, 1946. Distinct miliary nodulation throughout the left lung. Only questionable nodulation is seen on the right. The patient is asymptomatic. (*B*) July, 1952. The pattern has changed to one of marked bilateral fibrosis. Emphysema is present at both bases. The patient has developed chronic pulmonary symptoms. (*C*) November, 1953. Large bullae are apparent in the upper third of the left lung and in the axillary region of the right lung. (*D*) October, 1954. Bullous tranformation has now also occurred in the left apex. The patient has severe respiratory insufficiency. Superior mediastinal widening has persisted throughout the illness.

gress; instead there appeared to be slow progression with seeming transformation of part of the fibrotic process into additional cysts. In 1 of the 2 cases presenting a miliary pattern, fibrosis supervened before bullae developed (Fig. 2*B*).

The ultimate appearance was often quite characteristic (Fig. 2, *C* and *D*). There was widespread involvement of the lungs with strand-like or ill-defined densities, honeycombed with large or small thin-walled cavities having the typical appearance of bullae in 7 cases and of cystic bronchiectasis in 2. In addition—and an important part of the picture—there was definite enlargement of the hili and/or the superior mediastinum.

Bronchograms were available in 2 cases and both revealed moderately extensive cystic bronchiectasis; 1 showed, in addition, crowding of the bronchi in both upper lobes, indicating collapse or fibrosis.

Five of the 9 cases died, and autopsy was performed in 3. In each autopsied case widespread pulmonary fibrosis, bullous emphysema, and bronchiectasis were found. Two showed miliary noncaseating sarcoid tubercles in the lungs and other viscera. In the third, microscopic evidence of sarcoid was lacking at autopsy, but the diagnosis had been established earlier by biopsy of a peripheral lymph node.

A multicystic roentgen pattern may be encountered in tuberculosis, histoplasmosis, progressive bullous emphysema, pneumoconiosis, and many other diseases. However, the association of this pattern with enlarged tracheobronchial lymph nodes has, in a number of cases in the present series, enabled us to suggest the diagnosis of sarcoid, which subsequently proved to be correct. Earlier roentgenograms showing more typical roentgen features of sarcoidosis, and clinical and laboratory evidence consistent with this disorder are, of course, very helpful in the differential diagnosis.

The sequential roentgen changes appear to substantiate the viewpoint that the cystic changes result from pulmonary fibrosis. While it is realized that the roentgen identification of fibrosis is not always reliable, the criteria used in the present cases seem adequate. These included strand-like densities, persistence of the shadows for periods often in excess of a year, and retraction of fissures, hili, and mediastinal structures.

The seriousness of this multicystic complication of sarcoidosis is evident from the fact that 5 of the 9 cases died of the disease and 2 others were in poor state when last seen. The multicystic pattern, then, represents a late manifestation of the disease.

HISTOPLASMOSIS

A variety of pulmonary roentgen findings have been described in histoplasmosis.[6,17,21,23,27,44,45] For the most part, these findings have fallen into three main categories: (1) an acute disseminated form with miliary or small patchy densities diffusely distributed throughout the lungs—sometimes encountered in epidemics;[1,13,31] (2) a localized pneumonic form; and (3) the solitary nodule ("coin" lesion, histoplasmoma). Each of these types may be associated with tracheobronchial lymph node enlargement of variable degree and extent. At times, lymph node enlargement may be the only roentgen manifestation of the disease.

The healing phase is often characterized by the deposition of calcium in the pulmonary lesions and lymph nodes. The persistent spherical focus, or histoplasmoma, is also generally considered to be a healed or inactive lesion, and frequently shows calcification.

These well-known roentgen manifestations are by no means the only ones encountered in the disease. Other patterns do occur, some fairly frequently. Indeed, the diagnosis can sometimes be suspected from the roentgen appearance. A discussion of several of these less familiar patterns follows.

Fibrocavitary Histoplasmosis. This form of the disease has been considered rare. However, Furcolow and Brasher[18] found a number of cases in a Missouri tuberculosis

sanatorium, and other examples are being reported with increasing frequency.[25,37,39,49]

The condition has been seen chiefly among elderly males who have been admitted to tuberculosis hospitals with a mistaken diagnosis of chronic fibroid tuberculosis. The patients are chronically ill with the usual signs and symptoms of a long-standing pulmonary infection.

The chest roentgenogram reveals fibrotic involvement of one or both upper lobes, often with hilar elevation and displacement of mediastinal structures. Cavitation is a prominent feature, the cavities often being quite large and multiple. The pulmonary lesions may remain stationary, but more often they slowly progress to involve much of the lung parenchyma, with a fatal termination.

The sputum generally contains H. capsulatum. The skin and complement-fixation tests are usually positve. Coexistence with active tuberculosis,[18,30] malignant lymphoma,[21,40] and other fungus diseases is not rare.

Our experience with fibrocavitary histoplasmosis is based on 5 proved cases, 3 of which have been included in the paper by Bronson and Schwarz.[6] In each the diagnosis was first established in a tuberculosis sanatorium by the demonstration of H. capsulatum in the sputum. The average age of the patients was fifty-seven years, and all were white males.

The admission roentgenograms revealed widespread involvement, bilateral in 3 and unilateral in 2. In each instance at least one upper lobe was affected and in 2 an entire lung was involved. An irregular dense infiltrate with retraction of the hilar and mediastinal shadows and extensive cavitation was present in all cases (Fig. 3).

In 3 cases the lesions progressed, in 1 no change occurred over a two year period, while in 1 considerable regression was noted. The rate of roentgen change varied but was usually slow, a matter of months rather than of weeks. Two patients died, 2 are alive with active disease, and 1 improved strikingly during a ten month hospital stay but failed to return for a follow-up roentgenogram.

Fig. 3. Fibrocavitary histoplasmosis. Huge cavities are present in the upper two-thirds of the left lung. A small amount of infiltrate is seen in the right apex. The upper mediastinal structures are retracted to the left. Sputum positive for H. capsulatum. Fatal outcome.

The clinical and roentgen manifestations of fibrocavitary histoplasmosis may be simulated by other fungus diseases, tuberculosis, and certain bacterial infections, such as chronic Friedländer's pneumonia. Ordinarily the extensive cavitation associated with fibrocavitary lesions in these conditions promotes expectoration of sputum containing large numbers of the causative organism. Thus, appropriate sputum studies usually will disclose the correct diagnosis. This applies particularly in the case of tuberculosis. Therefore, any patient with a pulmonary roentgen pattern indicating fibrocavitary lesions and with sputum examinations which persistently fail to show tubercle bacilli should be studied thoroughly for histoplasmosis.

Enlarging Histoplasmoma. As stated earlier, pulmonary histoplasmomas are generally regarded as inactive lesions. However, we have seen 2 proved cases in which slow but definite enlargement took place over a period of several years (Fig. 4). In each instance the nodule contained calcium, strongly indicating a benign lesion, but the evidence of growth led to

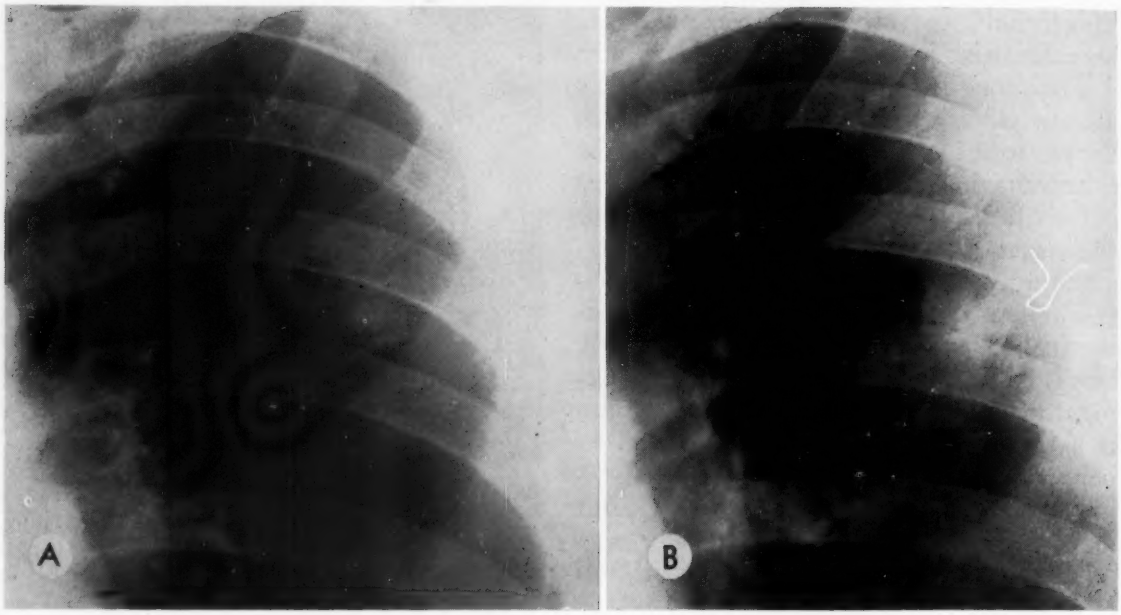

FIG. 4. Enlarging histoplasmoma. (*A*) October, 1954. A nodule with central calcification is present in the left third interspace. It measures 1.4 cm. in diameter. (*B*) February, 1957. The nodule now measures 2 cm. in diameter. Resected specimen contained H. capsulatum.

surgical resection. In both cases H. capsulatum was demonstrated in profusion in tissue sections, although the organism did not grow on suitable culture media. Notwithstanding these 2 cases, the histoplasmoma must be an exceedingly bland lesion since rarely, if ever, do new nodules or infiltrations develop in association with them.

Histoplasmic Broncholithiasis. The overwhelming majority of broncholiths are attributable to erosion into a bronchus by calcific foci from lesions of the lung parenchyma or tracheobronchial lymph nodes. It is generally assumed that healed or quiescent tuberculosis is the cause of these calcific foci.[15] However, in an area in which histoplasmosis is endemic, this disease, rather than tuberculosis, might be implicated.

There have been 2 recent cases in Cincinnati in which H. capsulatum has been demonstrated in tissue sections of broncholiths (Fig. 5). These are believed to be the first examples of histoplasmic broncholithiasis reported.[4] The demonstration of H. capsulatum in the only 2 instances in which a broncholith was studied for this organism lends support to our growing belief that histoplasmosis, rather than tuberculosis, is the underlying cause of most cases of broncholithiasis in this area.

Traction Diverticulum of the Esophagus. A similar case can be made for a histoplasmic rather than a tuberculous background for traction diverticula of the esophagus. A sequel of long-standing inflammation of lymph nodes, diverticula of this nature result from traction by cicatrix in and about the involved lymph nodes. Figure 6 illustrates the development of a traction diverticulum following an acute episode of intrathoracic lymphadenitis, possibly caused by histoplasmosis. We have also seen an almost identical occurrence in association with a fungus, probably Cryptococcus neoformans (Torula).[10]

Multiple Histoplasmomas. We have had the opportunity of following 5 asymptomatic patients in whom many large nodules, measuring up to 3 cm. in diameter, were demonstrated roentgenographically (Fig. 7). In each case one or more of the nodules appeared to contain calcium. On prolonged

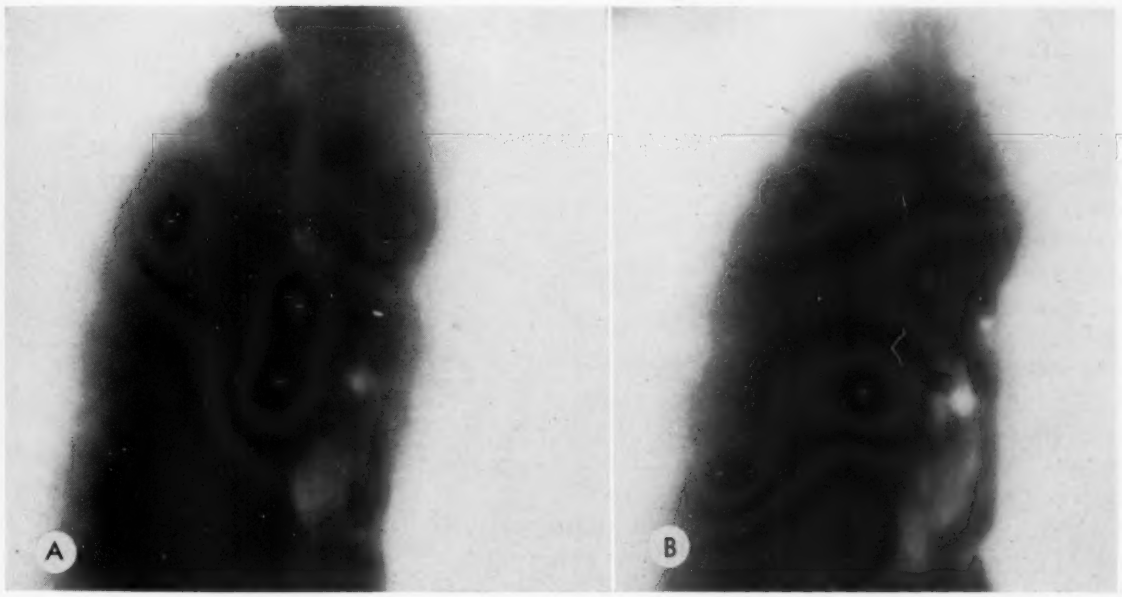

Fig. 5. Histoplasmic broncholithiasis. (*A*) September 20, 1956. Laminagram of right upper lobe. An infiltrate extends upward from the right hilus. Two fragments of calcium are seen at the proximal margin of the infiltrate. (*B*) October 10, 1956. Laminagram at same level. The lateral calcific fragment is no longer present and the infiltrate has regressed. H. capsulatum was found in the expectorated broncholith

observation the nodules remained stationary in 3 cases and became smaller in 2. In none of the 5 was a definitive diagnosis established.

The lack of progressive enlargement of the lesions and the continued well-being of the patients over long periods of observation exclude metastatic neoplasm from consideration. Sarcoidosis is ruled out on the basis of the calcification which appears in some of the nodules. The possibility of the rare nodular form of tuberculosis cannot be eliminated but in 2 cases the tuberculin test was negative. Multiple benign tumors, hamartomas, and other fungus diseases must also be considered in differential diagnosis.

The positive histoplasmin skin test in all the cases, the high correlation between pulmonary calcification and histoplasmosis in this section of the country,[16,47] and the similarity of the roentgen characteristics of the individual lesions to those of proved solitary histoplasmomas make it seem reasonable to suspect that at least some of these cases represent histoplasmosis.

Other Possible Sequelae of Histoplasmosis. For reasons similar to those expressed above, histoplasmosis is a likely frequent underlying cause of the so-called middle lobe syndrome and of chronic superior vena caval obstruction. In each instance fibrocalcific lymphadenitis and perilymphadenitis involve adjacent structures. These may, on the one hand, constrict a bronchus and, on the other, compress the superior vena cava. We have seen no proved instance of chronic bronchial obstruction resulting from histoplasmic lymphadenitis. However, in a patient recently operated upon at the Cincinnati General Hospital for superior vena caval obstruction, H. capsulatum was demonstrated in a lung lesion adjacent to a large granulomatous lymph node which impinged upon the superior vena cava. The lymph node itself did not contain the organism.

The variety of chest roentgen patterns seen in histoplasmosis is increasing as experience with this disease accumulates. The resemblance between these patterns and those of tuberculosis is often striking. In an endemic area, therefore, histoplasmosis must be given serious consideration in all

FIG. 6. Esophageal traction diverticulum resulting from mediastinal lymphadenitis possibly of histoplasmic etiology. (*A* and *B*) February, 1954. Posteroanterior and left oblique spot roentgenograms showing a defect on the right anterior wall of the esophagus. (*C* and *D*) August, 1954. Similar views. The defect has been replaced by a diverticulum.

cases in which a diagnosis of pulmonary tuberculosis is suspected but unconfirmed.

NONINFECTIOUS NECROTIZING GRANULOMATOSIS

In 1936 and 1939, Wegener[53,54] reported 3 cases with a severe destructive granulomatous rhinitis, a peculiar necrotizing granulomatous process in the upper respiratory tract and sometimes in the lungs, focal glomerulonephritis, and a more or less generalized angiitis resembling periarteritis nodosa. The nasal and facial manifestations of Wegener's cases were similar to those of a long-known condition reported under various names, including lethal granuloma of the midline facial structures, granuloma gangraenescens, and malignant granuloma.[5] Some authors consider the two diseases closely related and have suggested that lethal granuloma constitutes the localized form of Wegener's syndrome.[19,20]

In 1951, Churg and Strauss[7] reported a group of cases with severe asthma, fever,

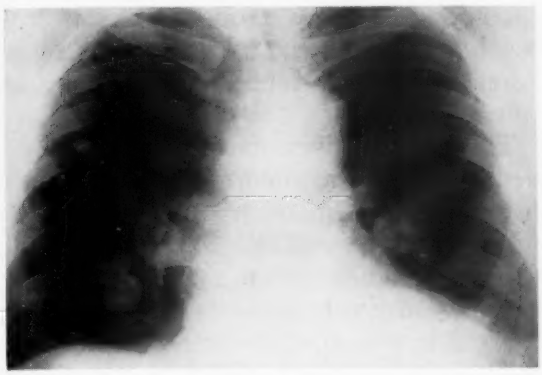

Fig. 7. Possible multiple histoplasmomas. There appears to be central calcification in several of the nodules. No change in appearance has been demonstrated over a period of three years. The patient is asymptomatic.

and eosinophilia, associated with necroitzing angiitis and extravascular granulomas in various organs. They called this condition allergic angiitis and granulomatosis. Its close similarity to Wegener's syndrome was soon recognized.[14,20]

Thus, there exists a group of diseases bearing a resemblance to each other, particularly in regard to the presence of necrotizing granulomas and angiitis, yet showing certain differences. Many cases of Wegener's syndrome, lethal granuloma, and Churg and Strauss's granulomatosis do not present the entire classic gamut of features. Moreover, cases have been encountered which represent varying combinations of these conditions.[19,20]

Cases falling into this group of diseases have been reported under many titles in addition to those already mentioned, including atypical periarteritis nodosa, giant cell granuloma, granuloma with periarteritis nodosa, and allergic granuloma. The need for a generic name is, therefore, apparent. Fienberg[14] recommended the term *pathergic granulomatosis* to indicate that a sensitivity phenomenon is involved in the pathogenesis. Since the etiology is still unknown, the more descriptive term *noninfectious necrotizing granulomatosis* is preferred in this presentation.

Clinical Findings. Our experience with noninfectious necrotizing granulomatosis is based on 6 autopsied cases, 5 representing Churg and Strauss's granulomatosis and 1 Wegener's syndrome The disease affects adults beyond the age of thirty almost exclusively; there is no racial or sex preponderance. The average age of the present cases was fifty-eight years. Four were male and 2 female; 4 were white and 2 were Negroes. Systemic symptoms, such as fever, weight loss, anemia, and leukocytosis were common, being present in 5 of the 6 cases. Allergic manifestations, including eosinophilia, commonly occur in the Churg and Strauss type, but were present in only 2 of the cases, including the patient with Wegener's syndrome.

When present, involvement of the face and upper respiratory tract is quite striking. Severe destruction of the midline facial structures, perforation of the nasal septum, and ulcerations and granulomas in the oral cavity, paranasal sinuses, and other portions of the upper respiratory tract occur.[5,24] The lesions may remain confined entirely to these locations, with no systemic involvement, in which case death occurs in six to twenty-four months, usually from inanition or hemorrhage. None of the patients presented here had facial lesions, but one of the Churg and Strauss group had sinusitis, perforation of the nasal septum, and ulcerated, crusted intranasal lesions.

When the lungs are involved, such symptoms as chest pain, cough, hemoptysis, night sweats, asthmatic episodes, and dyspnea are common. Four of the 6 patients exhibited respiratory symptoms, but only in 2 of these were they attributable to pulmonary granulomas, as determined pathologically. Clinical and laboratory evidence of renal involvement, including uremia, often occurs late in the course of the disease. These features were present in 4 of the cases. Indolent ulcerations of the skin are not uncommon and may, on occasion, be the earliest and most striking feature of the disease, as in the patient with Wegener's granuloma, the only example of cutaneous involvement in the series. Since almost any organ may be affected, clinical

manifestations referable to many systems of the body have been reported, and occurred in 2 of the present cases.

The average duration of the disease from time of onset of symptoms to fatal termination is said to be about six months, although individual patients may live for several years. The longest survival among the present cases was fifteen months. It is generally assumed that the disease is uniformly fatal—at least this has proved to be true in all the reported cases, the present series being no exception.

Roentgen Findings. When the facial structures are involved there is usually roentgen evidence of severe sinusitis with destruction of a sinus wall, the nasal septum, or other portions of the facial skeleton. None of the present cases had roentgen studies of this region.

A variety of chest roentgen findings have been reported in the literature.[3,8,9,19,26,28,29,32,41,42,46,48,53,54,55] Although the chest roentgenogram may remain normal throughout the course of the illness, nonspecific changes, such as bronchopneumonia and edema, as well as findings specifically attributable to the pulmonary granulomas, are common. The latter most often consist of solitary or multiple nodular densities or localized infiltrates. The nodules are poorly defined and often coexist with the infiltrations so that the distinction between the two types of shadows is obviously not clear-cut. The lesions vary from 1 to 8 cm. in greatest diameter and, when multiple, are seldom numerous. Evidence of cavitation in some of the lesions is often present, the cavities being small and irregular in outline. Other roentgen manifestations, rarely encountered, include pulmonary collapse, disseminated miliary lesions, bronchiectasis, and small pleural effusions. The pulmonary lesions may remain stationary or enlarge, or new densities may appear. Rarely, regression or even complete clearing may occur.

In the present series 2 cases had normal chest roentgenograms, 1 had bilateral pleural effusion and recurrent bouts of pulmonary edema, and 1 developed bilateral basal bronchopneumonia. In none of these were granulomas found in the lung at autopsy.

The remaining 2 cases demonstrated striking roentgen abnormalities which at autopsy were directly attributable to granulomatous involvement. In 1, the patient with the Wegener type of lesion in whom marked soft tissue destruction of the left thigh was the presenting complaint, the admission chest roentgenogram was normal. Seven months later a large area of infiltration containing multiple cavities was found in the right lung and a poorly circumscribed nodule with a radiolucent center in the left (Fig. 8). Two weeks later, shortly before death, the infiltrate on the right had enlarged and a left pleural effusion appeared.

In the other case, a patient with the Churg and Strauss syndrome who presented extensive peripheral lymph node enlargement and hepato-splenomegaly and in whom an incorrect diagnosis of Hodgkin's disease had been made, the initial roentgenogram showed moderate bilateral hilar and slight mediastinal lymph node enlargement. One year later a chest roent-

FIG. 8. Noninfectious necrotizing granulomatosis, Wegener's type. Recumbent chest roentgenogram. A large ill-defined area of infiltration with multiple cavities is seen in the central zone of the right lung and a solitary nodule with central cavitation is present on the left. Arrows indicate the cavities. (Reproduced with permission from *Radiology*.[12])

genogram revealed marked tracheobronchial lymph node enlargement, partial collapse of the right middle lobe, infiltrate adjacent to the right and left heart borders, and a small right pleural effusion.[12] The striking involvement of the lymphatic system shown by this patient is very unusual in this disease.

Pathologic Findings. As stated previously the two basic pathologic features of noninfectious necrotizing granulomatosis are (1) the aseptic granulomas and (2) the striking vascular lesions.

The granulomatous aspects are well-depicted in the lungs. Grossly, the lesions usually consist of multiple, fairly well-circumscribed nodules, the largest sometimes more than 5 cm. in diameter, often with necrotic centers. Histologically, the granulomas reveal a central coagulative necrosis surrounded by a zone of inflammatory cells in which eosinophils may predominate. Similar granulomas may occur in other structures, particularly in the upper respiratory passages, skin, spleen, lymph nodes, and kidneys.[20,29]

The vascular alteration consists of localized necrotizing inflammation of both arteries and veins, with fibrinoid changes. The angiitis may be widely disseminated, the lungs, kidneys, and spleen being most commonly affected. In some instances the vascular lesions are confined to the areas immediately adjacent to the granulomas, and sometimes they are even limited to one organ.

In the present series vascular involvement was widespread in 5 cases and confined to the kidneys in 1. Extravascular granulomas were widely distributed in 4 cases, limited to the kidneys in 1, and confined to the spleen in 1.

The histologic appearance of necrotizing granulomatosis is a striking one. There are microscopic differences between the Wegener and the Churg and Strauss types and between the vascular changes in these conditions and those of periarteritis nodosa. However, a detailed pathologic discussion is beyond the scope of this presentation. Suffice it to say that the histologic differences between these entities are not great, and most contemporary authors consider them related diseases.[56]

In the differential diagnosis many conditions must be given consideration, including malignant neoplasm, specific infectious granulomatous disorders such as tuberculosis and fungus disease, sarcoidosis, pemphigus and other dermatologic diseases, Loeffler's syndrome, and periarteritis nodosa. The clinical diagnosis of noninfectious necrotizing granulomatosis depends on the presence of several of the following manifestations: an allergic background, destructive granulomatous lesions of the face, skin, and upper respiratory structures, eosinophilia, evidence of renal disease, and the roentgen signs described above. One roentgen pattern is of particular importance in differential diagnosis, that of multiple poorly defined nodules or infiltrates with central cavitation. This pattern is unusual in other diseases and should bring to mind the diagnosis of necrotizing granulomatosis. Once this diagnosis is considered, steps can then be taken toward microscopic verification.

SUMMARY AND CONCLUSIONS

This report deals with three types of pulmonary granuloma: sarcoidosis, histoplasmosis, and noninfectious necrotizing granulomatosis. An attempt has been made to select material and to emphasize aspects of these diseases which are not familiar to many roentgenologists.

Under the heading of sarcoidosis is a description of two interesting roentgen patterns, one consisting of multiple large nodules, the other of multiple cysts. The subject of histoplasmosis is discussed with particular reference to the fibrocavitary form and to two unusual manifestations of the histoplasmoma—growth and multiplicity. Evidence is presented to indicate that in the endemic area broncholithiasis, esophageal traction diverticulum, "middle lobe syndrome," and chronic superior vena caval obstruction may represent late se-

quelae of chronic histoplasmic tracheobronchial lymphadenitis. Finally, under the generic term *noninfectious necrotizing granulomatosis*, the inter-relationships between Wegener's syndrome, lethal granuloma, and the Churg and Strauss allergic angiitis and granulomatosis are discussed and the clinical, pathologic, and roentgenologic findings in this group of related diseases are described and illustrated.

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ROENTGEN ASPECTS OF BLASTOMYCOSIS

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NORTH American blastomycosis, more commonly called blastomycosis, is a chronic infectious disease caused by the organism *Blastomyces dermatitidis*. The disease is characterized by the formation of suppurative, as well as granulomatous, lesions in any part of the body. The skin, lungs, and skeletal system are involved most frequently.[13] Gilchrist and Stokes, in 1894, described a case of cutaneous blastomycosis and identified the causative organism. Wohler and Montgomery, in 1902, reported the first case of the systemic form of the disease.[13]

Martin and Smith[8,9] emphasized that North American blastomycosis may exist in two forms, cutaneous and systemic. The cutaneous type presents as a chronic or subacute ulcerating process. The systemic type is characterized by pulmonary infection with widespread distribution of lesions in the subcutaneous tissues and skeletal system, less frequently in the internal viscera, and at times in the central nervous system.[13] Some investigators doubt that there are two distinct forms of the disease, and believe that it is simply a matter of the extent of involvement. Patients with pulmonary involvement usually develop cutaneous lesions. Many patients with so-called cutaneous forms of the disease have abnormal roentgen findings in the lungs.[4,11]

The systemic form usually begins insidiously with symptoms suggestive of respiratory infection. The patient may have a low-grade fever, cough, and chest pain. As the pulmonary lesion progresses, the patient may develop malaise, sweats and hemoptysis.[13] Dissemination of the fungus usually occurs by the hematogenous route.[11] The patients with blastomycosis of bone may present themselves because of a chronic draining sinus of the subcutaneous or deep tissues.[6]

The diagnosis of blastomycosis is made by direct smear and culture of the exudate from the cutaneous lesions, or the organism may be identified by examining the sputum. In some cases biopsy of skin or bone may be necessary. The diagnostic value of skin test and complement fixation is uncertain.

The roentgenographic findings in pulmonary blastomycosis are not uniform or characteristic. In fact, the infection may manifest itself in the lungs as disseminated miliary granulomas, homogeneous consolidation, solitary or multiple nodular densities, abscesses, or fibrotic lesions.[15] Hilar or mediastinal lymph node enlargement, as well as pleural involvement, may be seen.[5]

Osseous blastomycotic lesions are usually in cancellous bone and are predominantly destructive with little or no bone production. There is usually a sharply demarcated punched-out area seen on the roentgenogram.[2] Sometimes, the lesion may be manifested by a diffuse osteomyelitis, and occasionally there may be only periostitis.[10]

The roentgen findings of 5 patients with systemic blastomycosis with follow-up roentgenograms form the basis for this report.

REPORT OF CASES

CASE 1. M.H., a sixty-three year old colored female, was admitted to the hospital on April 19, 1955. The patient had an ulceration on the posterior aspect of the calf of the leg which had been present for three months. There was considerable pain and induration around the ulcer. Four weeks before admission, she had been hospitalized elsewhere and the lesion was incised. She received large doses of antibiotics with no improvement. One week before admission she developed a similar lesion on the medial aspect of the right arm. There were no pulmonary symptoms. Biopsy of the leg ulcer showed organisms morphologically identified as *Blastomyces dermatitidis*. Roentgen examination of the chest disclosed a homogeneous area of increased density adjacent to the left hilus (Fig. 1*A*).

The sputum smear showed yeast-like cells

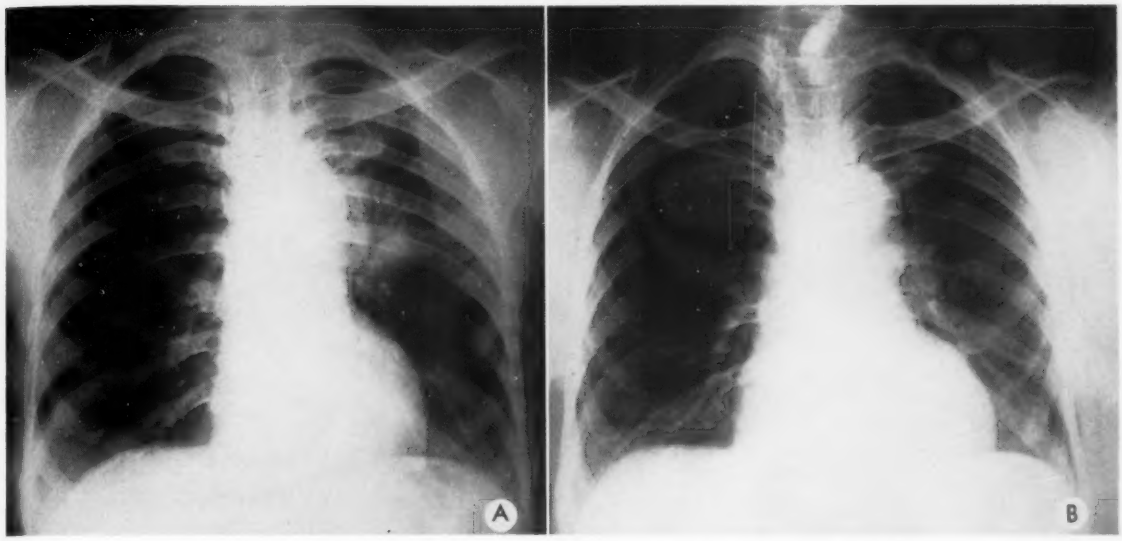

Fig. 1. Case 1. (*A*) April 19, 1955. Dense shadow adjacent to left hilus.
(*B*) May 29, 1956. Complete resolution of lesion.

identified as *Blastomyces dermatitidis*. Fungus culture of the sputum was done and *Blastomyces dermatitidis* organisms were isolated. The patient was started on stilbamidine which was changed to 2-hydroxystilbamidine when it became available.

The patient was dismissed, and was not seen for fourteen months when she was asked to report for check-up. The chest roentgenogram at this time showed complete clearing of the lesion in the left lung (Fig. 1*B*). The patient had gained thirty pounds and was asymptomatic.

Comment. This case represents the so-called cutaneous type. However, on a routine roentgenogram of the chest there was a pulmonary lesion which apparently was not causing any symptoms. Both the pulmonary lesion and the skin lesions responded to stilbamidine therapy.

Case II. R.B., a twenty-eight year old white male, was admitted to the hospital on April 16, 1954, with chest pain and fever. The patient had been in good health until three weeks before admission. There was no improvement after ten days of tetracycline therapy. The chest roentgenogram showed consolidation in the right lung base (Fig. 2*A*). Bronchoscopy was essentially negative. Culture of the bronchial washings was positive for *Blastomyces dermatitidis*. The patient was placed on 2-hydroxystilbamidine until the available supply was exhausted. He was then given stilbamidine. The elevation of temperature subsided, and there was much subjective improvement.

A chest roentgenogram four months later on September 2, 1954, showed practically complete resolution of the consolidated area in the right lower lobe (Fig. 2*B*). The patient was asymptomatic at this time.

Comment. This case apparently represents acute pulmonary blastomycosis as described by Bonoff.[3] The acute pulmonary case is benign as a rule; however, the patient may run a rapid downhill course and die.[14]

Case III. W.E.S., a thirty-six year old white male, was seen on April 3, 1953, with a chief complaint of pain around the left knee. The roentgenogram of the knee at this time was negative. The chest roentgenogram on April 3, 1953, showed multiple linear shadows extending from the left hilus to the lateral chest wall. There was also extensive miliary infiltration in the right mid lung field (Fig. 3*A*).

The patient left the hospital, but returned on May 8, 1953, with an elevated temperature and an increase in the size of the left thigh. The roentgen examination showed periosteal proliferation along the mid-shaft of the femur (Fig. 4*A*). An incision and drainage of the right thigh and femur were done. There were several ounces of thick pus. A bone biopsy showed blastomycosis organisms. The patient weighed 122 pounds at this time. He was given stilbamidine

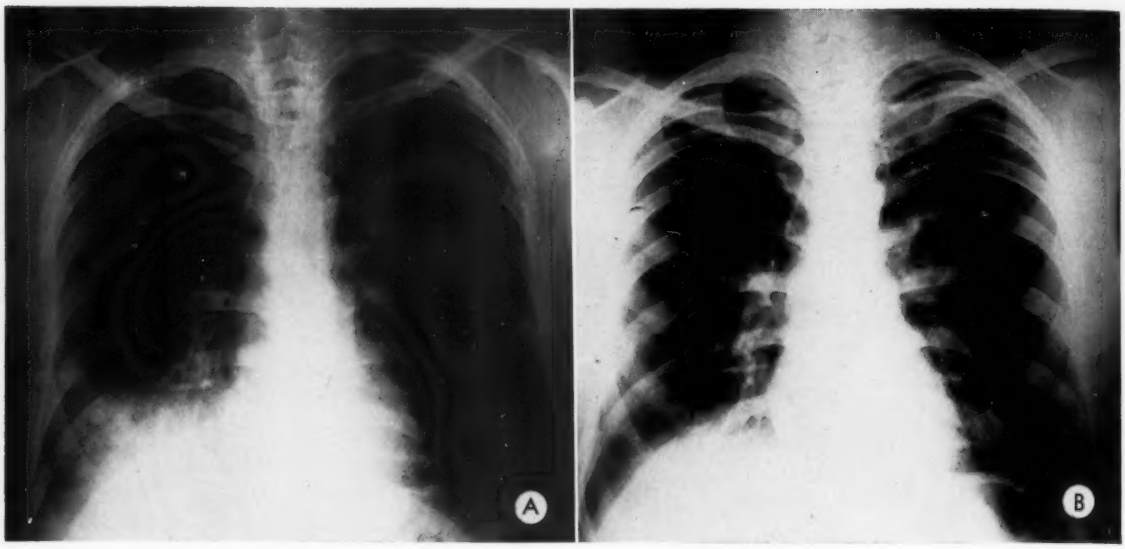

FIG. 2. Case II. (*A*) April 16, 1954. Consolidation in right lung base. (*B*) September 2, 1954. Marked clearing of the lesion.

and improved rapidly. On March 10, 1954, the patient weighed 185 pounds and he has stayed in this range. Serial roentgenograms of the chest showed gradual fibrosis of the parenchymal lesions. The last one on September 6, 1956, revealed the granular appearance in the right lung to be less. The lesions in the left lung were fibrotic (Fig. 3*B*). The roentgenogram of the femur showed thickened cortex along the shaft (Fig. 4*B*).

Comment. The lesions in the lung were assumed to be blastomycotic after the bone lesion was proved definitely to be due to blastomycosis. There was good response of both lesions to stilbamidine therapy.

CASE IV. M.H.W., a fifty-year old white female, was admitted to the hospital on August 22, 1951, with a chief complaint of pain in her left hand for three months. There was swelling and ulceration between the first and second metacarpals. There was a purulent drainage from the ulceration. The roentgen examination of the left hand on this date revealed marked bone production along the shaft of the first metacarpal (Fig. 5*A*). A roentgenogram of the chest on the same date showed patchy infiltration in the left upper lobe with small cavitations. There was a similar smaller area in the right infraclavicular region, also with cavitation (Fig. 6*A*).

The sputum smear and sputum culture were

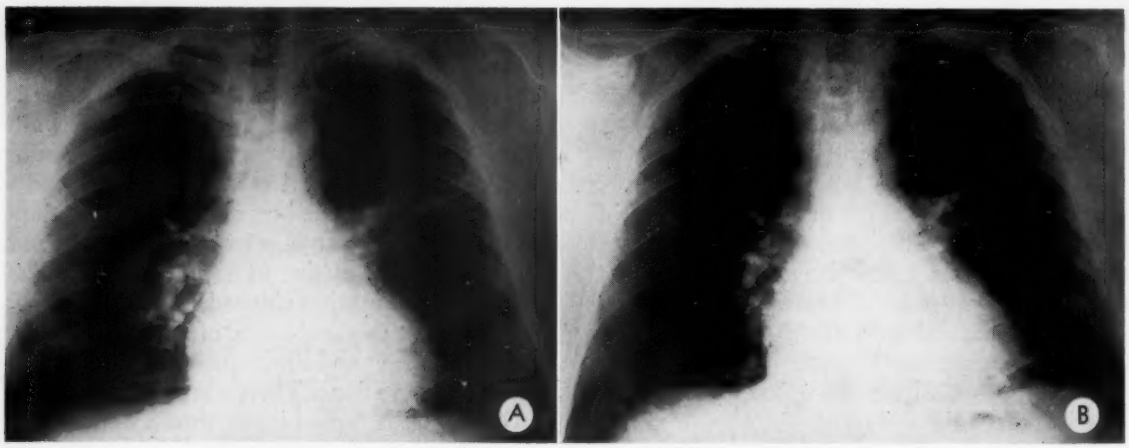

FIG. 3. Case III. (*A*) April 3, 1953. Linear densities in the left lung; small nodulations in the right lung. (*B*) September 6, 1956. Fibrotic strands in the left lung and diminution of the shadows in the right lung.

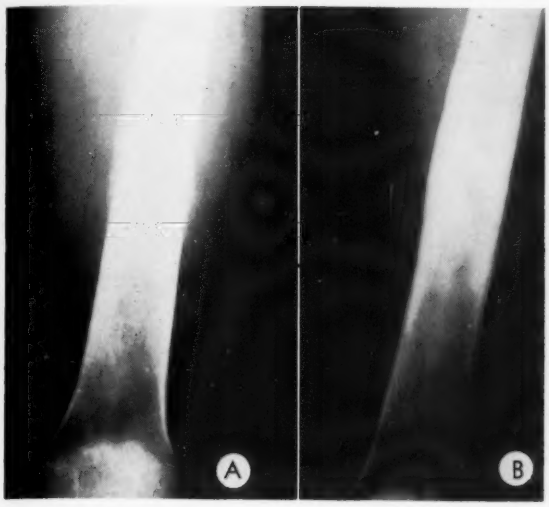

Fig. 4. Case III. (*A*) May 13, 1953. Periosteal reaction along the shaft of the femur. (*B*) September 6, 1956. Residual thickening of the cortex.

positive for *Blastomyces dermatitidis*. A smear of the exudate from the thumb also showed blastomycosis organisms.

Two weeks later the patient developed pain and swelling of the left leg anteriorly with sinus formation. A roentgenogram of the left tibia on September 4, 1951, showed two areas of irregular demineralization in the upper shaft anteriorly (Fig. 7*A*). The patient received a course of stilbamidine at this time. The sinus tract in the hand healed promptly.

On December 14, 1951, the roentgenogram of the chest showed moderate resolution of the infiltration and disappearance of the cavities. The roentgenogram of the hand on the same date revealed beginning healing of the lesion in the first metacarpal (Fig. 5*B*). The roentgenogram of the tibia demonstrated further destruction of bone in the two areas of the proximal shaft anteriorly (Fig. 7*B*).

On February 2, 1952, the patient was readmitted to the hospital because of the persistent draining sinus of the left leg. The roentgenogram showed progression of the destruction in the two areas. Saucerization of the two areas was done on February 5, 1952. The sputum was positive for blastomycosis organisms at this time. The patient received another course of stilbamidine.

Re-examination of the patient on September 5, 1952, revealed much subjective improvement. The chest roentgenogram showed fibrosis of the areas in the lungs (Fig. 6*B*). There was complete healing of the lesion in the first metacarpal (Fig. 5*C*). The operative defect in the tibia was evident, but there was no evidence of active disease (Fig. 7*C*). The patient has been

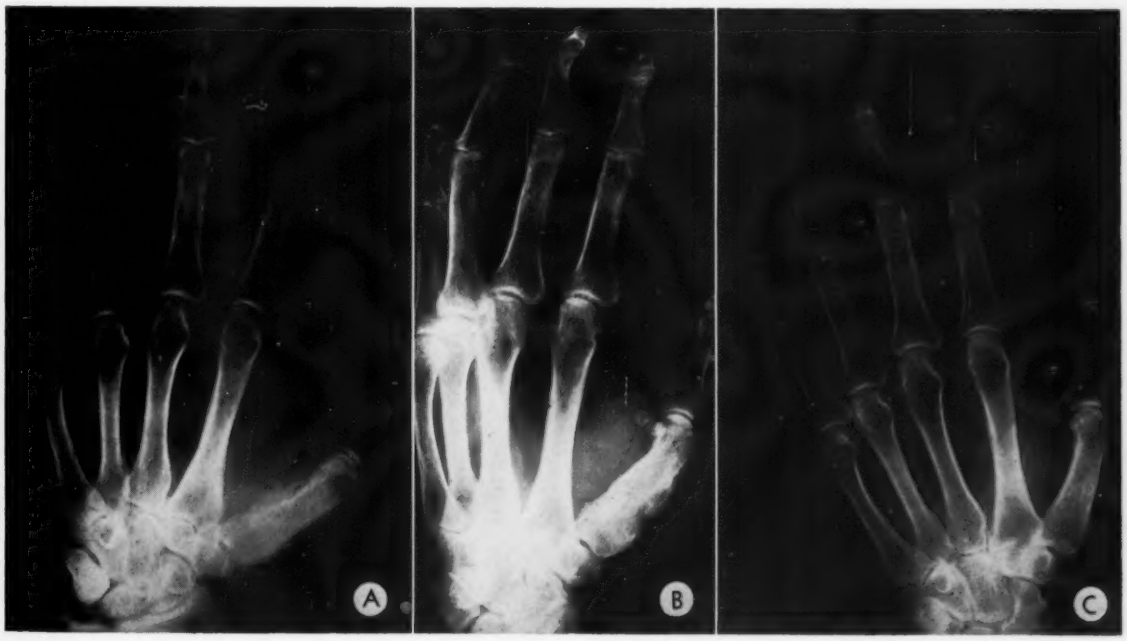

Fig. 5. Case IV. (*A*) August 22, 1951. Marked bone production along the shaft of the first metacarpal. (*B*) Four months later on December 14, 1951, beginning healing of the diseased bone. (*C*) One year later on September 5, 1952, complete healing.

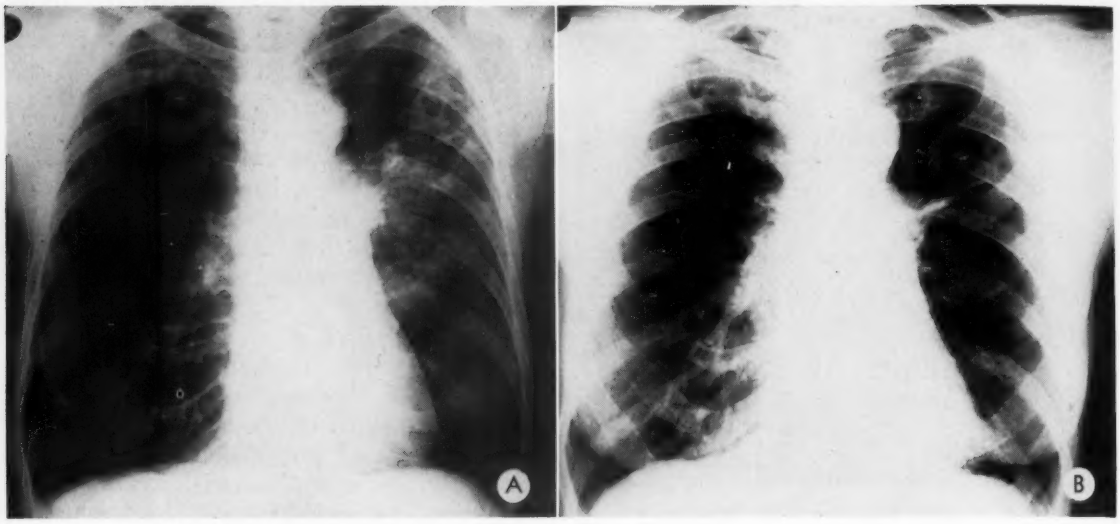

FIG. 6. Case IV. (*A*) August 22, 1951. Bilateral lung lesions with cavitations. (*B*) One year later, September 5, 1952, fibrotic lung lesions.

followed regularly. The fibrotic areas in the lungs have remained the same. There have been no new bone lesions.

Comment. This case illustrates another type of lung lesion with cavitation which is unusual. There were multiple bone lesions. One lesion responded to stilbamidine therapy while the other one progressed.

CASE V. A.A. was a forty-five year old white man with a chief complaint of a chronic draining sinus of the right hand. A roentgenogram of the hand on April 17, 1956, showed irregular demineralization of the shaft of the fourth metacarpal laterally with periosteal reaction along the medial aspect (Fig. 8*A*). A smear of the exudate showed *Blastomyces dermatitidis* organisms. A culture of the exudate was also positive for the same organism. A roentgenogram of the chest demonstrated a few calcified lesions around both hili. The patient was treated with 2-hydroxystilbamidine. A roentgenogram of the hand on June 11, 1956,

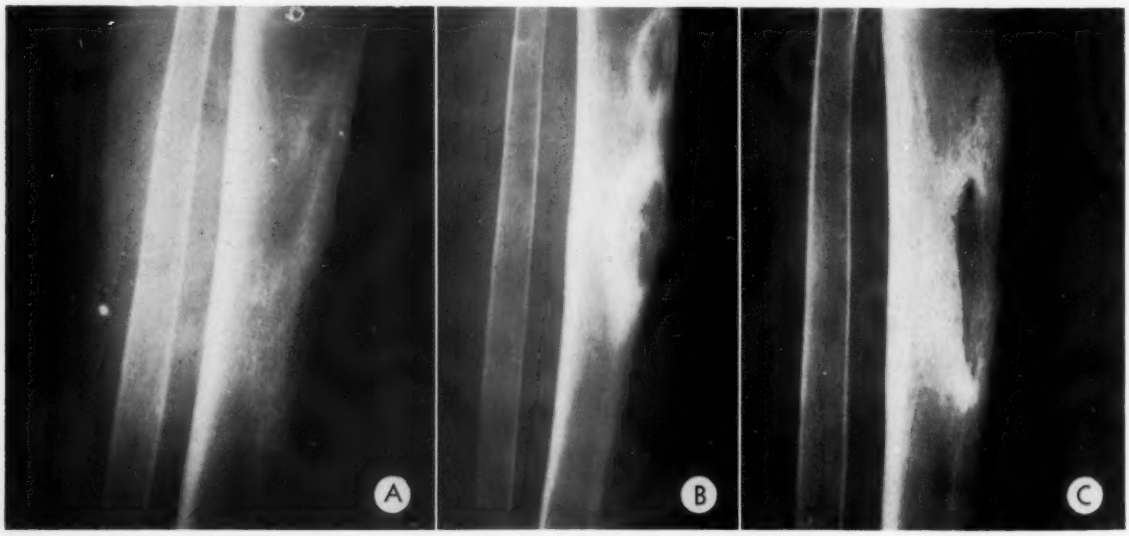

FIG. 7. Case IV. (*A*) September 4, 1951. Two areas of early bone destruction in the tibia anteriorly. (B) December 14, 1951. Progressive bone destruction. (*C*) September 5, 1952. Healed operative defect with no evidence of active disease.

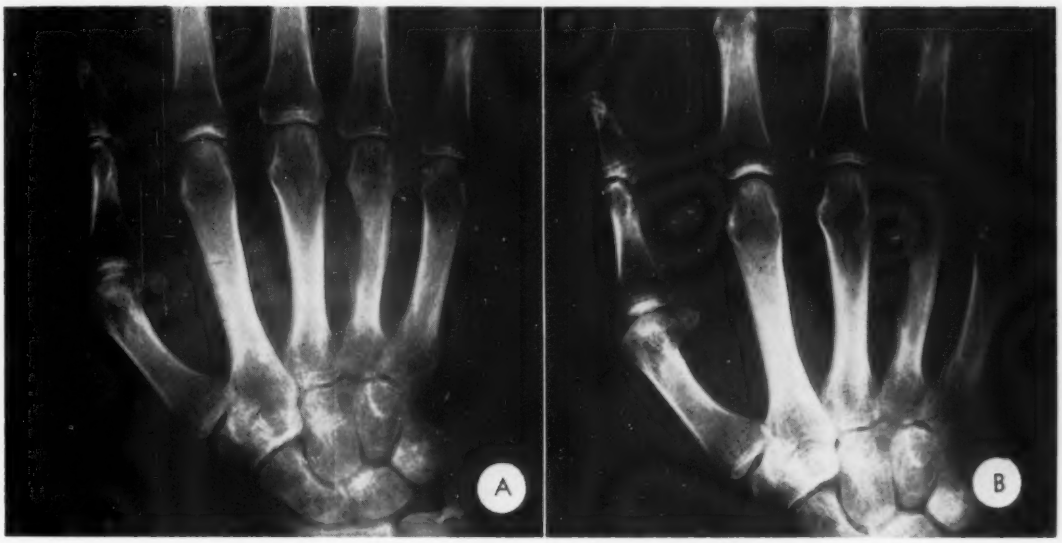

Fig. 8. Case v. (*A*) April 17, 1956. Lesion in fourth metacarpal. (*B*) Two months later on June 11, 1956, beginning healing of the bone lesion.

showed marked improvement of the lesion involving the fourth metacarpal (Fig. 8*B*).

DISCUSSION

From these cases it is evident that there is no characteristic pattern of pulmonary blastomycosis. Since the disease is a granuloma, it must be considered with and differentiated from tuberculosis, coccidioidomycosis, moniliasis, histoplasmosis, and torulosis.[1] One differential point mentioned in the literature is marked pleural involvement which is out of proportion to the parenchymal involvement.[5] The pleural lesion is usually manifest by localized thickening along the inner border of the thoracic wall rather than pleural effusion. Our cases did not show this finding.

The concept has been recently introduced that human blastomycosis exhibits a great tendency to heal regardless of its location.[1,11] Thus, there may be many unrecognized cases in which spontaneous recovery takes place, such as occurs with histoplasmosis and coccidioidomycosis. In other words, the patient may never be aware of having had the disease.

Our patients with blastomycosis involving the bone usually presented themselves because of a chronic draining sinus. Roentgenograms showed either bone destruction or periostitis.

The differential diagnosis includes tuberculosis and coccidioidomycosis.[15] Bone involvement with blastomycosis is characteristically near the end of the metaphysis and the joint is affected secondarily. In tuberculosis, the joint is involved early and by selection. Other differential considerations are metastatic tumor, myeloma, Ewing's tumor, syphilis, and sometimes bone cyst.[2,10] When the lesions are multiple, blastomycosis should always be considered.[10]

In any patient with bone involvement, a roentgenogram of the chest should be made, as this is considered to be the portal of entry.[4,15] Blastomycosis should be considered when a destructive bone lesion is found along with pulmonary or pleural involvement. The dermatologic manifestations are the most important clue to the diagnosis.

SUMMARY

Pulmonary blastomycosis may manifest itself by different patterns in the roentgenogram. The lesions may become stable or may resolve with stilbamidine therapy.

Blastomycosis involving bony structures

may appear on the roentgenogram as either a destructive process or as periostitis. Any patient with a chronic draining sinus exhibiting roentgen evidence of periostitis or a destructive lesion in the underlying bone should, therefore, have direct smears and cultures taken to exclude blastomycosis.

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Appreciation is extended to Dr. John Harvey for permission to use Case II.

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PULMONARY HISTIOCYTOSIS X (EOSINOPHILIC GRANULOMA)

REPORT OF A CASE

By J. PAGAN-CARLO, M.D.,* *and* TIMOTHY J. HALEY, M.D.†
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THE problem of the so-called eosinophilic granuloma, apparently primary in the lung, has been reviewed lately by Auld[2] and Arnett.[1] However, the variation of protean manifestations of this pathologic entity, labelled by Lichtenstein[6] as histiocytosis X, poses the question whether there is such a disease as primary eosinophilic granuloma of the lung. It is the purpose of this paper to emphasize the roentgenographic manifestations of this disease in the lungs and at the same time to stimulate consideration of the possibility that the reported cases of primary eosinophilic granuloma of the lung (pulmonary histiocytosis X) may simply represent what Farinacci, Jeffrey, and Lackey[4] call the "monosymptomatic expression of a disseminated process."

REPORT OF A CASE

W. F., aged fifty-nine, was admitted to the hospital for the first time on July 13, 1957, for a general examination because of an extensive fibronodular infiltration of both lungs, discovered on a routine physical check-up. At this time he was asymptomatic. There was a history of urinary infection four months previously but the patient apparently recovered completely. A chest roentgenogram made elsewhere one year prior to admission was reported as negative (Fig. 1).

The physical examination was essentially noncontributory. Laboratory studies included smears and cultures of several gastric washings and sputums, which were negative. The hemogram and urinalysis were normal. The blood chemistry for nonprotein nitrogen and glucose, the stool examination and the serology were negative. The skin tests with histoplasmin and purified protein derivative were positive, but complement fixation tests with blastomyces and histoplasma were negative.

The patient was discharged on July 17, 1957, with a diagnosis of diffuse pulmonary fibrosis of unknown etiology. On August 20, 1957, he was re-admitted for further investigation. At this time the physical findings remained unchanged. Repeated examinations of gastric washings and sputums were all negative.

Roentgen Findings. A chest roentgenogram revealed a rather extensive bilateral peribronchial infiltration of both lungs. The lung markings appeared beaded, thickened, and in some areas, nodular. Mild lobular emphysematous changes were present, with what appeared to be a small emphysematous bleb in the left apex. A fibrocalcific focus measuring about 8 mm. in diameter was present in the right upper

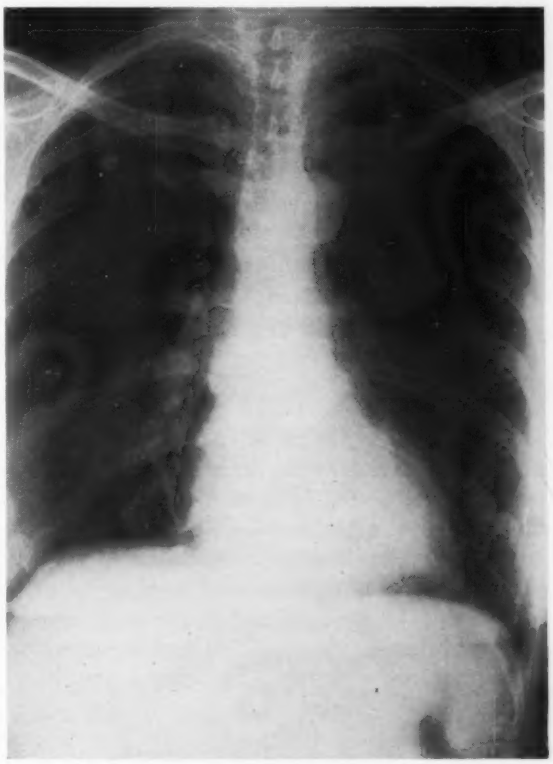

FIG. 1. Chest roentgenogram taken one year prior to patient's admission was reported as negative.

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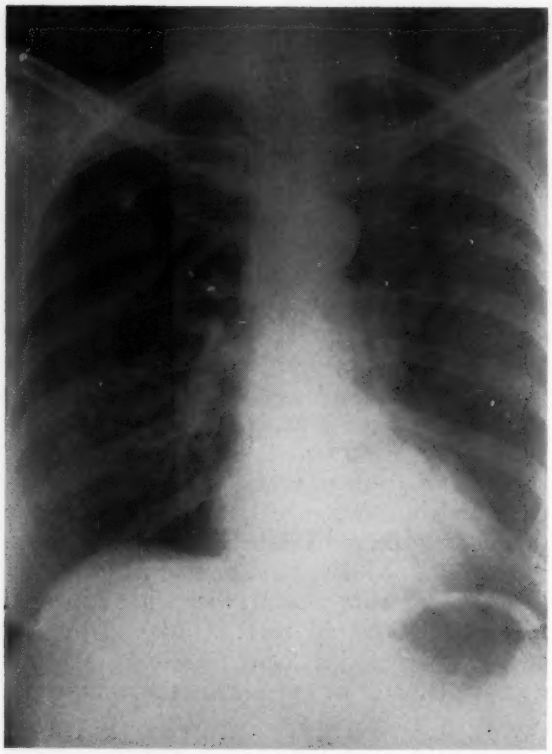

FIG. 2. Chest roentgenogram taken in August, 1957 showing extensive bilateral peribronchial infiltration in both lungs.

lobe at the level of the second rib anteriorly. The cardiac shadow was normal as to size and outline. No significant mediastinal lymph adenopathy was noted. There was no evidence of fluid accumulation in the chest and the bony rib cage appeared intact (Fig. 2).

On August 21, 1957, an exploratory left thoracotomy and lung biopsy were performed.

Operative Findings. The entire lung appeared and felt nodular, some of the nodules measuring up to 1.5 cm. in diameter. A few adhesions were present at the apex of the lung. The color of the lung was not unusual; the grayish nodularity and umbilication so frequently seen in pulmonary tuberculosis with miliary spread was absent. On section, one of the lymph nodes appeared dense, white and of hard consistency.

Microscopic Findings. Histologic study revealed small nodules composed of large numbers of eosinophils mixed with pale histiocytes. The pulmonary parenchyma showed interstitial fibrosis and areas of emphysema with thick fibrous alveolar septa. Some of the alveoli were filled with macrophages. The findings were considered diagnostic of eosinophilic granuloma of the lung (Fig. 3, *A* and *B*). Acid fast and Schiff stains were negative for organisms.

Treatment. The patient was started on corticosteroids (meticorten) and discharged on September 4, 1957. Subsequent follow-up by the attending physician in his private office revealed a marked improvement of the fibrosis in both lungs as demonstrated in subsequent chest roentgenograms taken in December, 1957 and February, 1958 (Fig. 4, *A* and *B*). A roentgenographic skeletal survey failed to reveal any evidence of osseous lesions.

DISCUSSION

Since Farinacci, Jeffrey, and Lackey[4] reported 2 cases of primary eosinophilic granuloma of the lung in 1951, Mazzitello,[8] Virshup and Goldman,[9] Auld,[2] and Arnett[1] have reported 11 additional cases. Others like May, Garfinkle, and Dugan,[7] and Grant and Ginsburg[5] have published cases complicated by extrapulmonary disease.

Engelbreth-Holm, Teilum and Christensen[3] divided the histopathologic process of what Lichenstein[6] calls histiocytosis X

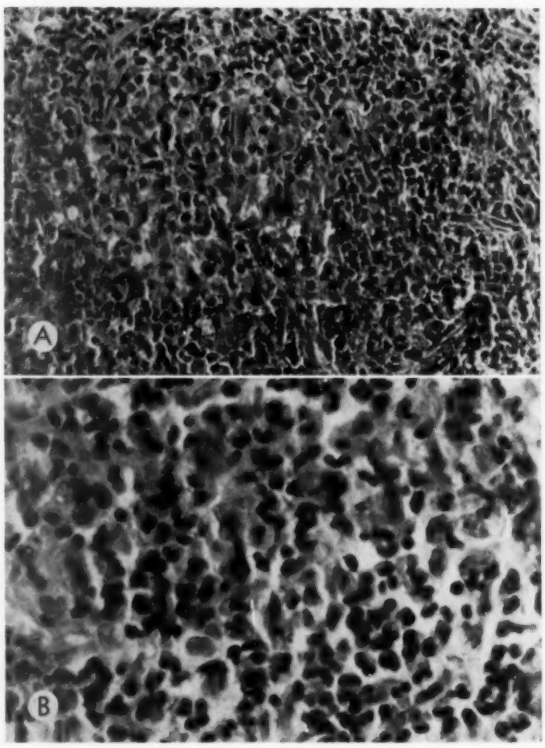

FIG. 3. (*A*) Photomicrograph showing syncytial masses of histiocytes with scattered eosinophils. (*B*) Same, larger magnification.

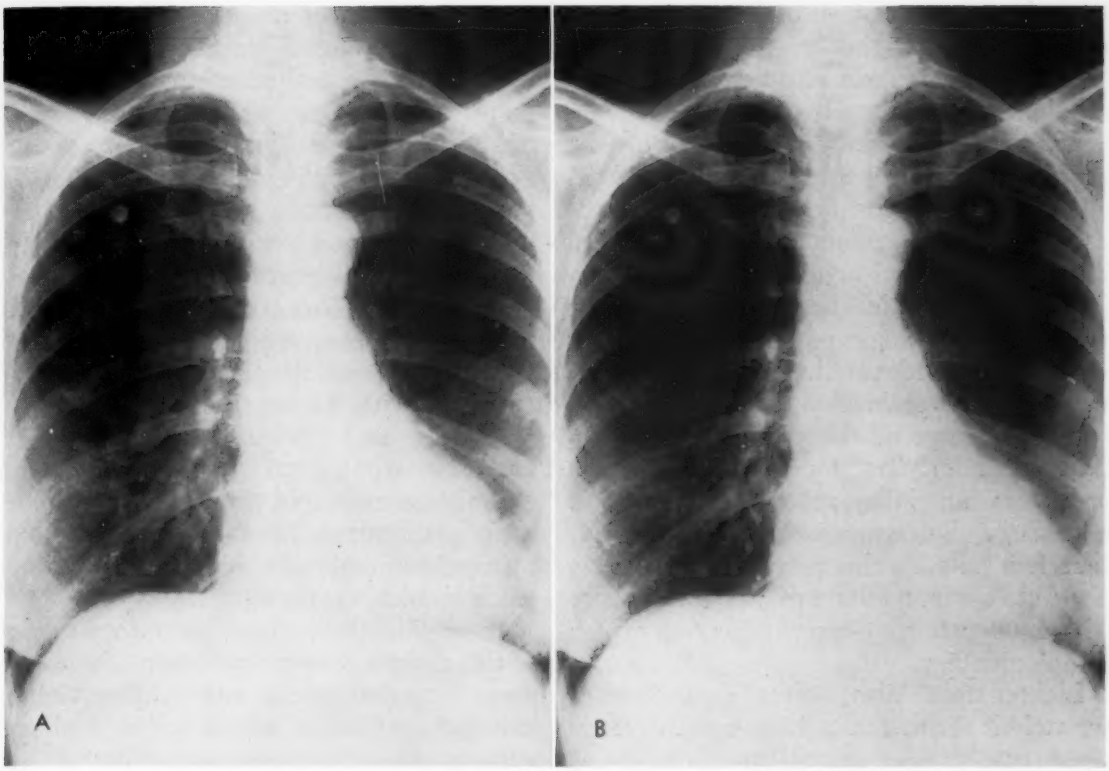

Fig. 4. Chest roentgenograms taken in December, 1957 (*A*) and February, 1958 (*B*) showing improvement of fibrosis in both lungs.

into four stages; namely, the hyperplastic or proliferative stage, the granulomatous stage, the xanthomatous stage, and the final or late stage of fibrosis. After correlating the gross and microscopic findings with the roentgenographic findings, Arnett[1] described two distinct roentgenographic patterns: (1) a discrete, soft, nodular infiltration and (2) an interstitial, reticulated infiltration. In our case, both patterns were present at one time.

The differential diagnosis of a diffuse fibronodular infiltration in the lungs includes such conditions as the pneumoconioses, disseminated or miliary tuberculosis, diffuse carcinomatosis, Boeck's sarcoid, the Hamman-Rich syndrome, polyarteritis nodosa, acute disseminated lupus erythematosus, etc. However, generally speaking, most of the patients with pulmonary histiocytosis X (eosinophilic granuloma) are not acutely ill and do not have the clinical and laboratory findings associated with many of the above mentioned conditions.

Auld,[2] in his excellent article on the pathology of eosinophilic granuloma of the lung, discusses a very interesting hypothesis as to the probable etiology or pathogenesis of this condition. He found certain common features as, for example, multiple granulomatous nodes, with heavy eosinophilic infiltration and interstitial inflammation. Acute arteriolitis with eosinophilic infiltration of the walls and, at times, acidophilic degeneration of the supporting tissues were also noted. The older lesions apparently undergo necrosis due to ischemia and the entire nodule may be extruded into the bronchus or bronchiole. In other instances fibrous tissue or scar may replace the nodule. Auld points out that the microscopic evidence, hence, indicates some type of hypersensitive state such as found in polyarteritis nodosa and Loeffler's syndrome. He further adds that improvement

of the process by the use of steroids tends to substantiate this theory and that the underlying allergen or antigen may be an inhalant.

Lichenstein's classic discussion of the problem in question apparently has cleared considerably the confusion with reference to eosinophilic granuloma, Letterer-Siwe disease, and Hand-Schüller-Christian syndrome; all of which he believes represent the same pathologic entity with different manifestations depending upon the severity, stage, dissemination, and, to a certain extent, the age of the patient. He states that the underlying process in all these disorders is an inflammatory histiocytosis stimulated by some unknown agent X, therefore he calls this process histiocytosis X. Auld's comments are not in conflict with Lichenstein's observations but rather complementary.

Lichtenstein[6] also states that "unfortunately it seems not to be generally recognized that diffuse pleural and interstitial pulmonary infiltration, leading eventually to fibrosis, honeycombing of the lungs and episodes of pneumothorax, is a rather common hallmark of chronic disseminated histiocytosis X (Schüller-Christian) at least in adults. Specifically it is not common for such cases to be regarded initially as instances of sarcoidosis." He points out the strong possibility, as yet not proved, that those cases presenting themselves only with the pulmonary manifestations of histiocytosis X will subsequently prove to be cases of chronic disseminated histiocytosis X and, hence, stresses the need for long term follow-up of all cases. Relatively little is known concerning comparable early lesions of disseminated histiocytosis X in internal or visceral sites other than the lungs, although their presence may be inferred from the distribution of lesions observed in cases which come to the autopsy table.

Virshup and Goldman[9] are convinced that the pathologic process in the lungs is the same as that found in the Letterer-Siwe disease and the Hand-Schüller-Christian syndrome. May and co-workers,[7] in reporting 3 cases of this disease with extrapulmonary involvement (skeletal), point out that the so-called eosinophilic granuloma of the lung or bone and Hand-Schüller-Christian syndrome are variants of the same pathologic process. Thus, it appears that the cases reported as primary eosinophilic granuloma of the lungs with no other demonstrable lesions elsewere may well have been cases of chronic disseminated disease, which at the time manifested itself primarily in the lungs. We tend to agree with Lichenstein, May and his colleagues, and Farinacci and co-workers, and classify pulmonary histiocytosis X as a chronic disseminated process with involvement predominantly of the lungs. A complete classification of histiocytosis X appears in Lichenstein's article.[6]

In view of the potentially serious nature of this disease, the examination of patients presenting themselves with a diffuse granulomatous or fibrotic infiltration in the lungs without subjective symptomatology should be general and extensive, with close cooperation between the radiologist, internist, pathologist, and surgeon. Grant and Ginsburg,[5] in reporting one case of pulmonary eosinophilic granuloma associated with diabetes insipidus, emphasize the importance of early pulmonary biopsy to determine the activity of the process and of immediate institution of therapy to prevent progression of the disease to the terminal stages of the honeycomb lung, severe fibrosis with emphysema, or the development of cor pulmonale. It appears that the basic procedure for proper diagnosis is an early pulmonary biopsy, with a concomitant exploration of the viscera or tissues accessible to biopsy and susceptible to histiocytosis such as peripheral lymph nodes, bone marrow, liver, etc. Pertinent laboratory studies and roentgenographic investigations to rule out the presence of other diseases capable of producing the same roentgenographic pattern in the lungs are, of course, indicated.

With regards to therapy we may say that some cases have cleared up spon-

taneously, others have been greatly benefited by roentgen therapy or by steroids. The success of therapy obviously depends on an early diagnosis and the stage or activity of the process, together with the presence or absence of long term involvement of vital organs. When the disease is in the active proliferative stage the use of steroids is indicated; in the later stages of the disease, with extensive pulmonary fibrosis and severe emphysema, direct therapy probably will be of little value other than supportive.

SUMMARY

A case of pulmonary histiocytosis X (eosinophilic granuloma) is presented. The histopathology and roentgen findings in this disease are discussed. A review of the literature points to the possibility that pulmonary histiocytosis X may be a "monosymptomatic expression of a chronic disseminated process," the etiology of which may be a hypersensitive state to an antigen, which in the case of primary pulmonary involvement may be an inhalant. It appears that corticosteroids are of definite therapeutic value in the early proliferative or granulomatous stage of the disease.

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The case presented in this manuscript was referred by Dr. Sydney Jacobs, attending physician and Chief, Department of Medicine, Touro Infirmary, New Orleans, Louisiana. We wish to acknowledge the invaluable help of Dr. A. Hertzog, Chief Pathologist, and of Dr. D. Bradburn, Associate Pathologist, Touro Infirmary, New Orleans, Louisiana. Roentgenograms are reproduced by courtesy of Dr. M. Teitelbaum, Radiologist, Prytania and Delachaise Streets, New Orleans, Louisiana.

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PULMONARY PARAGONIMIASIS*

AN EVALUATION OF ROENTGEN FINDINGS IN 38 POSITIVE SPUTUM PATIENTS IN AN ENDEMIC AREA IN THAILAND

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PULMONARY paragonimiasis is a disease caused by the presence within the lung parenchyma of the oriental lung fluke, *Paragonimus westermani.*

In a recent paper on pulmonary paragonimiasis, Yang *et al.*[16] of Taiwan, Formosa, reported that 88 out of 100 cases showing positive sputum also showed abnormal chest roentgen findings. The most prominent abnormality in their series was a "well defined nodule." "Ring shadows" were found in only 2 patients. There were 30 cases of pleurisy with and without effusion and 4 cases of spontaneous pneumothorax. Eight months after this article was published, Miller and Walter[8] pointed out that ring shadows are observed rather frequently in pulmonary paragonimiasis.

MATERIAL

In 1957, a survey for *Paragonimus* infestation was undertaken in a district of Saraburi Province, Thailand. A series of 140 persons with chronic cough as the chief complaint was examined. A positive sputum for ova of *Paragonimus westermani* was found in 38 cases. Chest roentgenograms were taken of these 38 patients and the lung findings studied.

This article presents an evaluation of the chest roentgen findings in the 38 positive sputum cases. As a whole, the incidence of abnormal shadows was as high as in the series of Yang and his co-workers, but they consisted mostly of ring shadows due to cystic cavities, and the burrows caused by the parasites could be clearly demonstrated by laminagraphy.

BACKGROUND INFORMATION

The first endemic area of *P. westermani* identified in Thailand[15] was the Nong Mu district in the southern part of Saraburi, lying about 100 kilometers north of Bangkok. This district, a hilly country with eight small streams, comprises mainly rice fields in which snails, crabs and shrimps are found in abundance. It is one of the practices of the population to eat raw or undercooked crabs or shrimps with spices and vinegar or lemon juice. The drinking water is taken from the unpurified streams. It has been suggested that *P. westermani* may have been introduced into this area by a number of Thai war veterans returning from Korea during the last five years. The environment of Nong Mu is particularly favorable to the life of the lung flukes.

CLINICAL FEATURES

Patients whose sputa were positive for *P. westermani* ova presented three cardinal symptoms: repeated blood-stained sputa, chronic cough and chest pain. One patient exhibited creeping subcutaneous eruptions on his face; another had cerebral symptoms. Only one had ova of the parasite demonstrated in the feces. The age distribution ranged from eleven to fifty-four years, the average being thirty years. The ratio between males and females was approximately 5 to 3. Symptoms were present over a period of from six months to five years. The average count of the eosinophils in the peripheral blood was 15 per cent. No pleural fluid was noted in any of the cases. Only 2 patients had intradermal tests for paragonimiasis, using the antigen* prepared from the adult worm. Both of these were positive.

* The antigen was kindly supplied by Professor B. G. MacGraith, School of Tropical Medicine, Liverpool, England.

* From the Departments of Radiology and Medicine, Siriraj Hospital and Medical School, Dhonburi, Thailand.

Table I

INCIDENCE OF ABNORMAL ROENTGEN FINDINGS IN THE CHEST

	Patients	Per Cent
Sputum positive for ova of *P. westermani*	38	100
Abnormal roentgen findings in the chest roentgenograms	36	95
Negative chest roentgenograms	2	5

ROENTGEN FINDINGS

Abnormal roentgen findings in the chest were encountered in 36 of the 38 cases studied (Table I). These were classified into four types: (1) Characteristic ring shadows; (2) opacities with poorly defined borders; (3) linear infiltration; and (4) pleural thickening. The changes varied according to the stage of the disease[9] and often there was an intermingling of several types on the same roentgenogram. By taking into consideration only the predominating type, the following relative figures were obtained: (1) Characteristic ring shadows, 20 cases; (2) opacities with poorly defined borders, 9 cases; (3) linear infiltration, 3 cases; and (4) pleural thickening, 4 cases (Table II). If the intermingled lesions were also counted the respective figures were: (1) 31 cases; (2) 18 cases; (3) 8 cases; and (4) 8 cases. In addition, calcifications were noted in 4 cases (Table III).

(1) *Ring Shadows.* As may be noted, the ring shadows were observed most frequently, occurring in 82 per cent of all cases. They represented the thin-walled borders of cystic cavities with a crescent-shaped opacity along one side of the border, resembling a solar eclipse during the corona stage. Usually they were multiple and aggregated, but, occasionally, a solitary lesion was also found. They ranged in size from 6 mm. to 4 or 5 cm. in diameter and therefore in the majority of instances were clearly visualized on the routine roentgenograms. In the obscure cases laminagraphy was of definite value in demonstrating the cystic cavities within ill-defined, irregular opacities or the associated linear infiltrations when these changes predominated.

Table II

PREDOMINATING TYPES OF ABNORMAL ROENTGEN FINDINGS IN THE CHEST

	Patients	Per Cent
Characteristic ring shadows	20	52
Opacities with poorly defined borders	9	24
Linear infiltration	3	8
Pleural thickening	4	10

Table III

INCIDENCE OF THE VARIOUS TYPES OF ABNORMAL ROENTGEN FINDINGS IN THE CHEST

	Patients	Per Cent
Characteristic ring shadows	31	82
Opacities with poorly defined borders	18	48
Linear infiltration	8	21
Pleural thickening	8	21
Calcifications	4	11

A study of the distribution of the cystic cavities within the chest revealed a definite preponderance of involvement of the right lung (Table IV).

Another important finding frequently associated with the ring shadows was the demonstration of tracts or burrows communicating with the adjacent cysts. The burrows appeared to run an irregular, tortuous course. Laminagrams revealed that their lumen measured as much as 5 mm. in diameter, compared to 2–3 mm. of the corresponding medium and small bronchial lumina which on bronchographic examination often appeared to be slightly narrowed.

Table IV

DISTRIBUTION OF THE LESIONS IN THE LUNGS

Location	Cases
Both lungs	6
Right lung, entire	13
Right lung, upper lobe	6
Left lung, entire	6
Left lung, upper lobe	5

Some of the bronchi were definitely displaced and showed irregularity and distortion, probably due to pressure of the solidified or fibrous lesions. Other bronchi were dilated or completely destroyed. Combined bronchography and laminagraphy disclosed that the tracts and burrows were separated from the opacified bronchi.

(2) *Opacities with Poorly Defined Borders.* These were observed in 48 per cent of all cases (Table III). Roentgenographically they appeared as a mottling or as nodular shadows of 3 to 4 cm. diameter. Some had a partially well defined border. They were usually present in the lower and peripheral lung fields, but occasionally involved the apices. Characteristic ring shadows were often noted within these opacities, and, when they were not, laminagrams were taken to reveal them. The configuration of the opacities, not infrequently, changed during the course of the disease. In general, the roentgen picture was suggestive of pneumonitis as is found in Loeffler's syndrome, gnathostomiasis or ascariasis. The latter two conditions are common in Thailand.

(3) *Linear Infiltration.* These shadows, resembling linear streaks seen in pulmonary fibrosis, were found in 21 per cent of all cases (Table III). They occurred mostly in the periphery of the lower lung fields and were associated with typical ring shadows. The degree of surrounding pneumonitis was minimal, apparently because of the chronicity of the course.

(4) *Pleural Manifestations.* Pleural thickening, likewise, occurred in 21 per cent of all cases (Table III), usually along the lower lateral chest wall. It seems that the adult worm has a tendency to travel along the pleural planes; *i.e.*, on the surface of the diaphragm, over the visceral pleura into the interlobar fissures and then along the intrapleural fissures, where they provoke a reactive process, suggestive of interlobar fissure pleuritis. This was seen in a number of cases, appearing on the roentgenograms as small, streaky shadows leading from the lateral chest wall towards the opacities in the lung parenchyma.

It may be mentioned that there were no cases in our series with spontaneous pneumothorax or an appreciable amount of fluid in the pleural cavity.

Since roentgen abnormalities were encountered in 95 per cent of our patients having positive *P. westermani* sputa, it is evident that a roentgenologic examination of the chest is one of the best means of establishing the definitive diagnosis, estimating the extent of the disease and observing its subsequent course. A chest roentgen examination should also be utilized as a screening procedure.

DIFFERENTIAL DIAGNOSIS

Pulmonary Tuberculosis. Differentiation of pulmonary paragonimiasis from pulmonary tuberculosis with caseating cavities constitutes the most difficult problem. However, the cystic cavities produced by the lung flukes exhibit a rather characteristic opacity along a part of the wall, the remainder of the wall appearing clean-cut with very minimal, if any, perifocal reaction. They vary in size from a few millimeters to several centimeters in diameter, are located atypically in all parts of the lung fields and often occur in groups of 10 to 15 with a tendency to coalesce. The area in which they are found or the neighboring area not infrequently contains tortuous tracts or tunnels, the diameters of which exceed those of the corresponding bronchi. The over-all picture is that of a burrowing process with formation of granulation tissue and cysts. Fibrosis is also evident. In our series of patients, there was no case of pleural effusion. The general constitution of the patient, as a rule, is not much disturbed unless pulmonary tuberculosis coexists or a secondary infection supervenes.

Bronchopneumonia. This can be ruled out easily by the presence of the cystic cavities and the benignity of the course of the pulmonary paragonimiasis.

Bronchiectasis. Paragonimiasis affects mostly the bronchioles. The encysting wall is fibrous and variable in thickness. It may be very thick and surround the worm com-

pletely. The encysted portion in an open case communicates with the small and medium bronchi and the patient is able to expectorate ova containing sputum. Unless secondary infection supervenes, it is unlikely that bronchiectasis will develop.

REPORT OF ILLUSTRATIVE CASES

Case I. This female, aged eighteen, was admitted to Siriraj Hospital in October, 1955, with the chief complaint of repeated blood-stained sputa for a period of two years. Eleven days prior to admission, she noticed a swelling on the right side of her face. The swelling was migratory and moved to form a nodule over her inner lip. The nodule eventually ruptured and a small worm was found in the discharge. The worm was still alive and moved about before she threw it away. The worm was said to be dark brown in color; its size was approximately that of an uncooked grain of rice. The patient had always lived in Saraburi. She liked to eat raw crabs, which had been salted overnight.

Physical Examination. The patient was a well nourished girl with very few constitutional symptoms. Her lower lip showed slight discoloration and an indurated area. Sputum examination revealed numerous operculated eggs of *Paragonimus westermani*. Acid fast bacilli were never demonstrated in repeated smears and cultures. Eosinophils and Charcot-Leyden crystals were also present in the sputum. Examination of the feces showed ova of *P. westermani*. Repeated differential blood cell counts gave 8–31 per cent eosinophils.

Treatment. The patient did not respond to hetrazan. Emetine was then given for nine days. The ova rapidly diminished in number and completely disappeared on the ninth day. Sulphadiazine was also given for the treatment of associated bronchitis. After a ten week period of hospitalization, at which time the symptoms subsided and *Paragonimus* eggs could no longer be found in the sputum, the patient was discharged.

Roentgen Findings. Chest roentgenograms demonstrated a spherical cyst-like cavity (Fig. 1), measuring about 10 mm. in diameter, in the medial left infraclavicular region. It contained no fluid and its wall was smooth and thin. A crescent-shaped, poorly defined opacity was present at the inferior portion of the border of the cyst. The right infraclavicular region also showed a small area of opacity in its medial portion. Through this opacity, a cavity, similar to that on the left side but smaller in size, was seen. There was no infiltration elsewhere in the lung fields. Fluoroscopy revealed the cavity of the left upper lobe to be in the posterior segment of the lung. Subsequent roentgenograms taken at intervals during the course of treatment showed no change in size and appearance of the cystic cavities in the upper lobes, although the pneumonitis which was present in some of the surrounding area soon disappeared. The latter change was transitory and similar to Loeffler's syndrome.

Fig. 1. Case I. Roentgenogram showing typical ring shadow in the left infraclavicular region. Note the corona effect at the medial portion of the wall of the cystic cavity.

Case II. This female, aged twenty-one, was admitted to Siriraj Hospital in July, 1957. The patient had lived in the Nong Mu district, Saraburi, from the time she was eight years old until the previous year, when she moved to Bangkok. Three years prior to admission she had repeated blood-stained sputa. The blood loss was variable in amount, producing anemia and, occasionally, shock. A roentgenogram of the chest was taken with negative results. The patient, thinking that she had contracted pulmonary tuberculosis, sought treatment from a private physician. In spite of the treatment, she

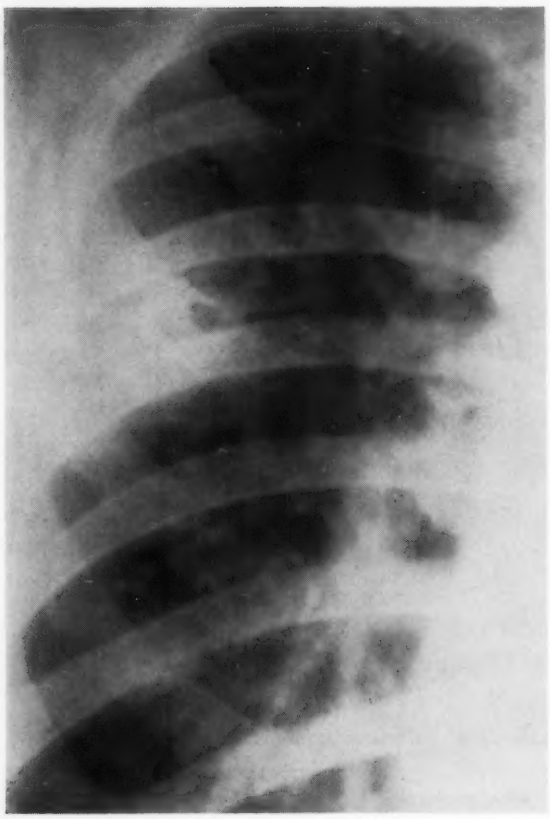

FIG. 2. Roentgenogram showing characteristic ring shadow in the right upper lung field.

FIG. 3. Roentgenogram showing typical cystic cavities in the right upper lobe.

FIG. 4. Roentgenogram showing opacities with poorly defined borders in both lung fields. Ring shadows are present in the right upper and left lower lung fields.

FIG. 5. Roentgenogram showing linear infiltration intermingled with ring shadows.

continued to have frequent chest pain. One month before admission she coughed up an amount of blood. Her symptoms became more aggravated and she was persuaded to come into our care.

Physical Examination. The patient appeared healthy but was slightly anemic. The chest was negative. Temperature, pulse and respiration were normal. Laboratory findings showed 7.0 gm. of Hb, 3.28 million red blood cells and 11,800 white blood cells per cu. mm. with a differential count of 34 per cent polymorphonuclears, 34 per cent lymphocytes and 32 per cent eosinophils. The sputum was positive for operculated ova of *P. westermani*. During the hospital course, although the patient coughed from time to time, the sputum contained less blood and, along with the feces, proved negative for ova of *P. westermani*. The patient was discharged free of symptoms on August 4, 1957.

Roentgen Findings. There was a localized opacity in the medial right upper lobe. Laminagrams of the region showed groups of small cystic cavities contiguous with each other. Tracts communicating with these cystic cavities were also demonstrated. A solitary cystic cavity was noted in the left base. Bronchograms revealed a slight degree of bronchial dilatation. Subsequent roentgenograms which were taken over a period of four months showed some clearing of the surrounding pneumonitis in the medial right upper lobe. The cystic cavity in the left base, however, did not show any appreciable change.

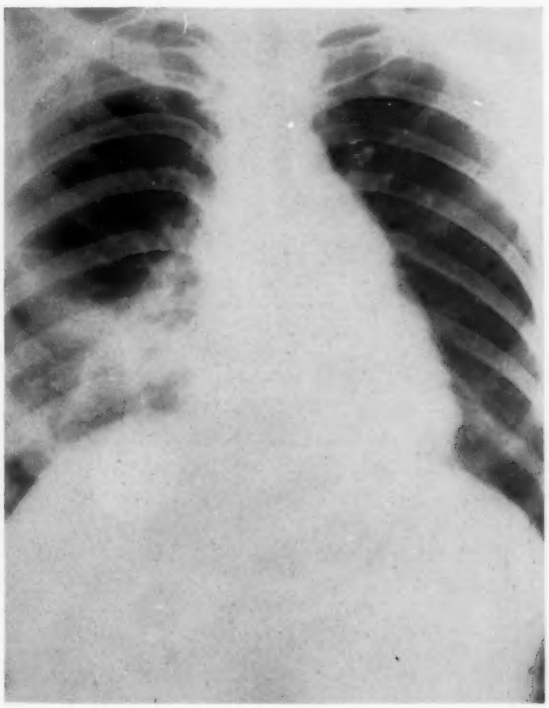

Fig. 6. Roentgenogram showing abnormalities which suggest interlobar pleuritis. Ring shadows are present in the right lower and left upper lobes.

Fig. 7. Roentgenogram showing calcifications. An appearance suggestive of ring shadows is present in the neighborhood of the nodule in the right upper lobe.

Case III. This male, aged twenty-one, also came from Saraburi, entering Siriraj Hospital in April, 1957. His chief complaint was chest pain of two years' duration. Three years previous to admission, he had had a chronic productive cough with mucous sputum. One year later, during military service, the cough became more frequent and the sputum became blood-stained. The amount of blood in the sputum was estimated to be about 200–300 cc. a day. The symptoms became more severe accompanied by general malaise, fatigue and an afternoon feverish sensation. The blood loss through expectoration sometimes reached one liter in amount at

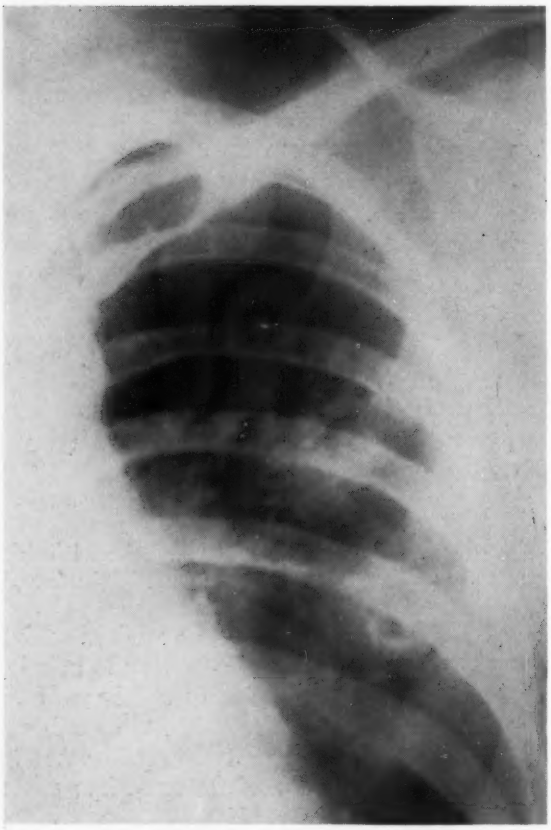

FIG. 8. Case III. Roentgenogram showing typical ring shadows in the left upper lung field.

a time. Six months prior to admission, the patient had developed an abdominal pain which was alleviated by massage. As in the previous 2 cases, the patient liked to eat crabs.

Physical Examination. The patient was a healthy appearing young man. The chest was negative. Laboratory findings were 10.9 gm. per cent of Hb, 4.39 million red blood cells and 7,800 white blood cells with a differential count of 68 per cent polymorphonuclears, 20 per cent lymphocytes, 2 per cent monocytes and 10 per cent eosinophils. Sputum was positive for operculated eggs of *P. westermani*. The feces were negative for ova. The ova in the sputum persisted after administration of chloroquin. Sulphadiazine was given for the treatment of secondary infection. Even so, cough and bloody sputum persisted.

Roentgen Findings. Examination of the chest revealed typical cystic cavities with surrounding pneumonitis. Laminagrams clearly demonstrated communicating tracts. The lesions were confined to the left upper lobe. Bronchograms with laminagraphic exposures showed that the typical ring shadows in this case communicated with the bronchi. The bronchi exhibited effects of pressure exerted by the lesions as evidenced by narrowing, irregularity and distortion. Some of the involved bronchi showed a slight degree of dilatation (Fig. 8, 9, 10 and 11).

DISCUSSION

An analysis of the abnormalities in the chest roentgenograms of 36 patients with pulmonary paragonimiasis of our series revealed that the ring shadows constituted the most common finding. This is in contrast with the observations of Yang *et al.*[16] who noted only 2 instances of ring shadows in 88 abnormal chest roentgenograms of 100 patients with a positive sputum.

Ross *et al.*[12] reported an interesting positive sputum case (a Chinese seaman from Nimpo near Shanghai) in which the ring shadows did not appear on the routine chest roentgenograms, but were demon-

FIG. 9. Case III. Laminagram demonstrating the burrow or tunnel leading to the contiguous cystic cavities.

strated by laminagraphy. They emphasized that the contiguous cavities are characteristic of the underlying pathology as shown by histologic examinatuion. In an illustration, they presented a cavity containing two adult flukes and stated that the cavities are produced directly by the flukes and are not due to bronchiectasis. This observation conforms with the pathogenesis of pulmonary paragonimiasis.[11]

Our findings strongly support the views of Ross *et al.* The ring shadows in our series were plainly visible on the routine chest roentgenograms. The tracts or tunnels connecting the cystic cavities were shown to lie separately from the dilated bronchi. Laminagraphy and bronchography with laminagraphic exposures confirmed these observations. The cystic cavities and burrowing resembled closely the type of infestation by the parasite, as described in standard textbooks on parasitology.[3,4,5,7]

Fig. 10. Case III. Bronchogram showing slight bronchial dilatation. There is communication of the bronchi with the cystic cavities in the left lung.

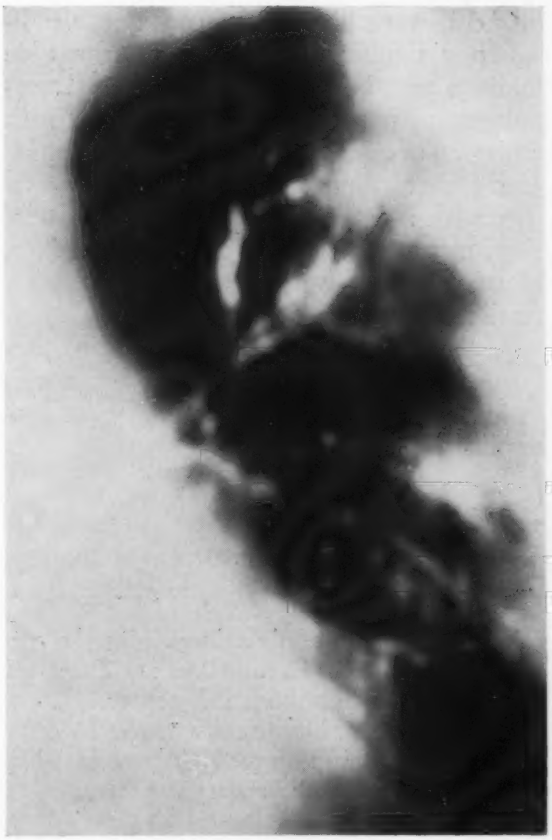

Fig. 11. Case III. Bronchogram with laminagraphic exposure showing the tunnel or burrow to be separate from the regional bronchi.

SUMMARY AND CONCLUSIONS

1. The chest roentgen findings were evaluated in 38 cases with positive sputa for ova of *P. westermani* from the endemic area of Saraburi. In 95 per cent of these cases the roentgenograms revealed the presence of lung flukes.

2. In contrast to the roentgen findings of Yang *et al.*[16] in Taiwan, the ring shadows were the most prominent feature in this series of patients, being present in 82 per cent of all cases. Existing connecting burrows were disclosed by laminagraphy.

3. The ring shadows were characteristic in appearance. The burrows which were demonstrated by laminagraphy and by bronchography with laminagraphic exposures to be separate from the regional

bronchi, were likewise of considerable diagnostic significance. These two roentgen findings may be considered as indicative of pulmonary paragonimiasis in patients with a history of endemic exposure.

4. Other abnormalities included opacities with indefinite borders, linear infiltration, pleural thickening and calcifications. However, these were usually seen in association with the typical ring shadows.

5. Roentgen examination should be performed for screening as well as diagnosis in an endemic area of pulmonary paragonimiasis.

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Vol. 81, No. 2

THROMBOEMBOLISM OF THE LUNGS*

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THE occurrence of one or several emboli in the lungs of persons who died of chronic debilitating disease, particularly cardiac decompensation, is a common observation. Many of these cases have no premonitory signs, such as thrombophlebitis, nor clinical symptoms from a lung infarct or embolus. The emboli do not contribute materially to the death of the patient and they might be classified as "incidental." Other emboli are of larger size or multiple and produce definite clinical symptoms resembling pneumonia, pleurisy, myocardial infarction, or even tumor, and have been named "contributory" emboli. A third category is known as the "massive" embolus blocking one or both branches of the pulmonary artery and leading rapidly to asphyxia and anoxemia. A similar effect is observed if showers of emboli block a wide field of pulmonary circulation leading to a fall in blood pressure, vasomotor collapse, acute cor pulmonale and shock. The incidence of detection of pulmonary emboli in patients dying in a general hospital has been given at about 10 per cent, while massive embolism has been found in 4 per cent of all hospital necropsies. It has been pointed out in several reports that the percentage of embolism and infarcts was considerably higher in patients in homes for the indigent and in paralytics.

Among the medical conditions contributing to thromboembolism are polycythemia vera, blood dyscrasias and malignancies, besides mitral stenosis and failure of the hypertensive heart. Immobilization of the lower extremities, extensive tissue resection, limb amputations and puerperium are responsible for the high percentage of the "surgical" emboli which usually occur within two weeks after injury or operation. Trauma either to muscle or skeleton, fractures and burns are among the causes of thromboembolism. Israel and Goldstein[12] have called attention to the common type of traumatic phlebitis which may be caused by repeated vein punctures, insertion of polyethylene tubes, injection of hypertonic solutions, and such medication as mercurial diuretics.

Cardiac surgery also contributes to the increase of thromboembolism which, in spite of prophylactic measures and vein ligation, appears alarming to Ochsner.[18,19] The conversion of a fibrillating heart under quinidine results in a 4 per cent fatality rate due to embolism. The auricles themselves are not infrequently the place of development of thrombi, particularly in cases of arrythmia, where there is an incomplete and irregular contraction and emptying of the auricular appendages. Mural thrombi, either adhering to the meshwork of the trabeculae or free and friable, are found in mitral stenosis. They can be recognized on roentgenograms if calcified, or on the left side by a slightly irregular, immovable bulge of the appendage segment. Large "ball thrombi" might fill the entire auricle and block the venous inflow. They might be calcified, or pedunculated and interfere with the heart function to such a degree as to cause gangrene of extremities or of the tip of the nose; if less conspicuous they may cause spells of fainting and a fading and recovery of the pulse beat. There occur autochthonous thrombi, which remain at their place of origin and grow by apposition; these can be found in a pulmonary artery or in a branch of the abdominal vessels; finally, there are thrombi and emboli which migrate by long and devious ways through open septa and an incompletely closed foramen ovale.

Postoperative pneumonia, atelectasis or pleurisy often are manifestations of embolism or infarction and should, in the future,

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be more carefully evaluated as such. The veins of the calves of the legs have since Roessle been accepted as the most common extracardiac source of emboli, and Hampton and Castleman[9] have stated that they observed a very large percentage of pulmonary emboli arising from symptomless thrombosis of the deep veins of the leg, usually the popliteal. Other authors, however, have found a frequent involvement of the pelvic veins, the hypogastric vein, and the prostatic plexus. It is, perhaps, of significance that Ochsner, whose renowned institution is particularly geared to the recognition, prevention and treatment of emboli, found in about 40 per cent of his cases of fatal embolism no primary source of the embolus. He observed the largest number of pulmonary emboli occurring in patients from the gynecologic and obstetric service, and the next largest number from the urologic service. As roentgenologists we are impressed by the frequency with which phleboliths, as the outcome of pelvic blood stagnation, are noted on roentgenograms of the abdomen, lung emboli being found much more frequently in women than in men. There is, as Crane[4] pointed out, still considerable disagreement as to the most likely sites of origin of fatal pulmonary emboli. Circulatory stagnation, decreased muscular activity and depending leg position appear to be prime factors, as illustrated by the report of Simpson on 24 cases of "shelter death," of presumably healthy individuals who, during the air-raids in London, were sitting in cramped positions on low chairs with legs depending, and died from lung emboli without direct injury. Many of our prophylactic measures are based on the avoidance of clotting and stasis by early ambulation and anticoagulant therapy. It has been a surprise to find that early ambulation alone did not materially reduce the frequency of thromboembolism, and that the sitting posture did not decrease the stasis. More effective was the method of Wilkins and Stanton, who applied elastic stockings to their hospital patients to increase the linear velocity of blood flow in the deep veins with good success.

In our own observation the pulmonary embolus or infarct has frequently preceded the development or manifestation of thrombophlebitis and, in several instances, the primary location has not been detected. Clinical symptoms are most prominent in cases of suppurative thrombophlebitis because of severe pain. The thrombus, however, being fixed to the wall of the vessel, shows little tendency to embolus formation. In contrast, the *bland* thrombus, which lies loosely within the lumen, presents few symptoms, but is easily propagated.[26] This, then, might explain why so many of the early cases of peripheral phlebothrombosis escape even daily examination, and the chest roentgenogram sometimes demonstrates the earliest signs of embolic occurrence.

The radiologists have contributed much to the diagnostic recognition of this condition. The names of Westermark,[25] Hampton and Castleman,[9] Hanelin and Eyler[10] and others are connected with various phases of roentgenologic study and recognition of thromboembolism. Westermark particularly has called attention to the ischemic lung, in which the regular vascular pattern over an entire lung or a lung segment disappears as a consequence of pulmonary embolus, with resulting occlusion of a large branch or the mainstem of the pulmonary artery. The reliability of this sign has been questioned by some investigators who did not find it of diagnostic value.

REPORT OF CASES

CASE 1. A young woman, four weeks prior to hospital admission, exhibited a normal vascular pattern on the chest roentgenogram (Fig. 1). She was suddenly taken ill with severe dyspnea, chest pain, cough and bloody expectoration. Re-examination at the time of admission (Fig. 2) revealed absence of the peripheral vascularity, with cut-off of both pulmonary arteries. The heart had an unusual appearance, which might be referred to as "amphoric" (resembling a Greek urn). This picture was explained by the autopsy findings twenty-four hours after ad-

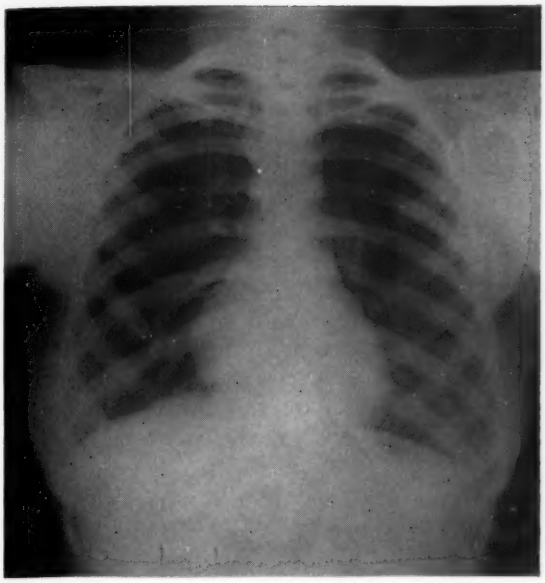

Fig. 1. Case 1. Normal chest roentgenogram of a twenty-nine year old housewife, taken four weeks before admission.

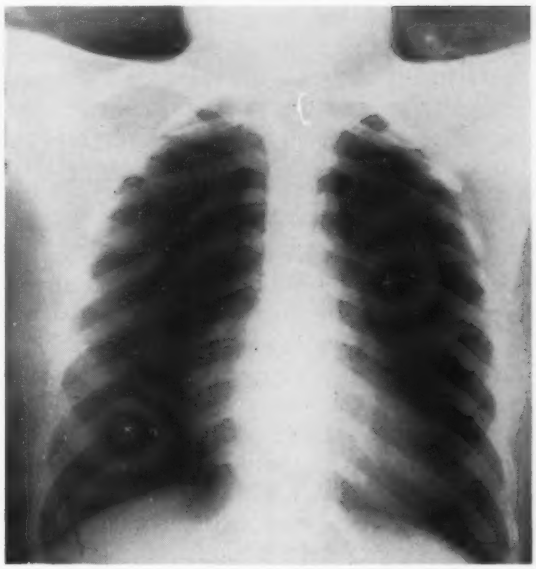

Fig. 2. Same patient as in Figure 1 admitted in acute respiratory distress. Sudden beginning of anxiety, chest pain, fever and cough with bloody expectoration. Sensorium clear. Note the disappearance of pulmonary arteries, transparency of lung and amphoric dilated heart. Thrombosis of mainstem of the pulmonary artery.

mission. Both main branches of the pulmonary artery were found occluded by recent and partially organized thrombi. The heart was that of a person with acute cor pulmonale. A partially organized thrombus was found in the right iliac vein.

Comment. Ischemia of the lung is rarely as well visualized as in Case 1 or in some of Westermark's[25] cases of complete occlusion of the main pulmonary arteries. Diseases commonly associated with pulmonary embolism are mitral stenosis with heart failure and, within the lungs, congestion, edema, hypostatic pneumonia and pleuritic reactions.

Suggestive of pulmonary embolism are the occurrence of sudden dyspnea after a relatively minor effort and a chest pain which, in intensity, resembles that of angina pectoris but is located behind the sternum and is more pleuritic in the case of infarction. Presenting symptoms are considerable restlessness and apprehension, an increasing cyanosis and, very frequently, hemoptysis. As the contributory cause of death, thromboembolism accounts for about 10 to 15 per cent of the cardiac cases.

Case II (Fig. 3) represents a different roentgenologic pattern. This was a forty-two year old female, with a past history of thrombophle-

Fig. 3. Case II. A forty-two year old female, operated on for papillary cystadenocarcinoma of the ovary, presenting complaints of severe chest pain on the fourth postoperative day. Expired on the ninth postoperative day. Note dilatation of both pulmonary arteries, dilatation of the superior vena cava and elevation of the right diaphragm. Bilateral pulmonary artery thrombosis with coagulation necrosis in the adjacent lung segments was found at autopsy.

bitis, admitted for surgery of an abdominal mass which proved to be a papillary cystadenocarcinoma of the right ovary. On the fourth postoperative day the patient suddenly complained of severe chest pain. After several such attacks she expired on the ninth postoperative day.

A chest roentgenogram had shown no evidence of Westermark's sign. Instead, a marked prominence of both pulmonary arteries, an elevated diaphragm on the right side, and a dilatation of the superior vena cava were visualized. Autopsy revealed the large and smaller branches of the pulmonary artery to be occluded by partially organized thrombi. There was coagulation necrosis in the adjacent lung segments, and the alveoli were filled with red blood cells. Both ovarian veins, as well as the right femoral vein were completely occluded by organized thrombi.

Comment. The underlying principle of roentgenologic recognition consists of a marked widening and prominence of the involved pulmonary artery and a sudden diminution of caliber, or complete cut-off, of the secondary branches beneath the usual massive embolus. This is well illustrated in the next case.

CASE III (Fig. 4, *A* and *B*) was a thirty-seven year old male with arrested tuberculosis and heart disease caused by pulmonary hypertension who was re-admitted because of swelling of the lower extremities and dyspnea of a few weeks' duration. The dyspnea increased rather suddenly five days later.

Autopsy revealed an organized large thrombus in the left pulmonary artery and a similar thrombus in the right atrium, besides pulmonary tuberculosis with a cavity in the right upper lobe. Right and left ventricular hypertrophy was present. The pulmonary artery in such cases is usually without pulsation and not compressible under Valsalva pressure exercise.

Comment. The roentgenographic appearance can well be called pathognomonic, as in the case from the Massachusetts General Hospital.[3] It permitted the radiologist to suggest immediately the correct diagnosis. Such cases have been further reported by Hanelin and Eyler,[10] and by Ball, Goodwin and Harrison[1] from the large material of the Middlesex Hospital in London, showing the almost aneurysmal widening of the blocked pulmonary artery and, at times, the tapering off of the peripheral

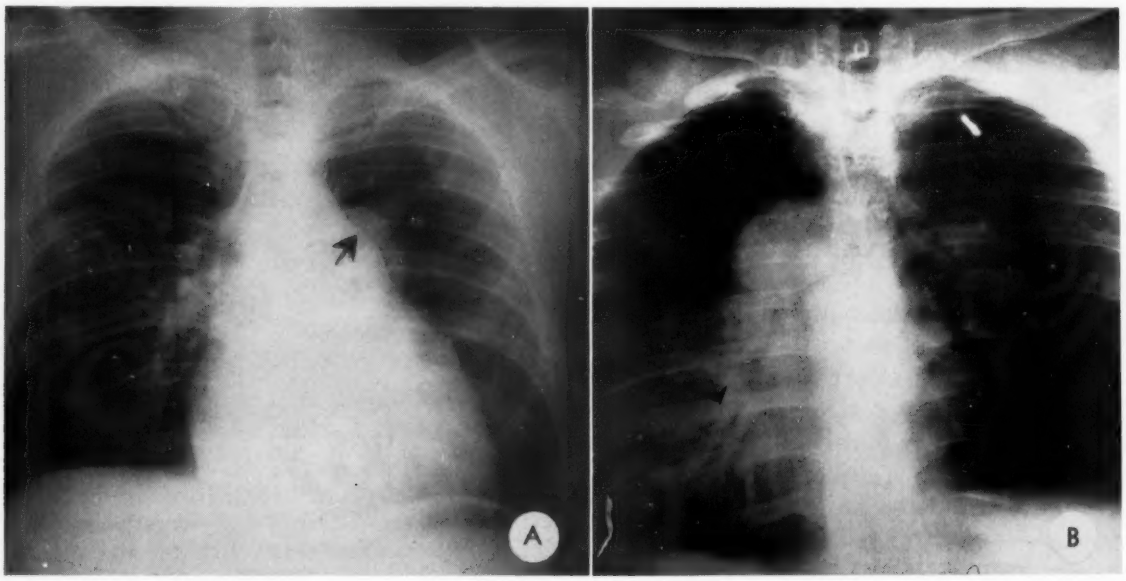

FIG. 4. Case III. (*A* and *B*) A thirty-seven year old male with arrested tuberculosis and heart disease caused by pulmonary hypertension. Ankle edema and dyspnea of four weeks' duration, which increased rather suddenly despite bed rest. The aneurysmal widening of the left pulmonary artery tapering off toward the basal segment is well visualized and explained by the large thrombus lodged within the left pulmonary artery. Note absence of pulmonary congestion and increased translucency of both lungs.

part with or without increased translucency.

Pulmonary emboli are one of the most frequent causes of acute cor pulmonale. Multiple and recurrent infarction leads to the development of chronic cor pulmonale. The dividing line between small and large pulmonary emboli should not be drawn too strictly as to their hemodynamic effect, since small emboli, while frequently silent, sometimes produce physiologic and clinical effects as striking as those of obstruction of the main pulmonary artery, even leading to sudden death.[5]

The development of cor pulmonale tends further to exaggerate the prominence of the pulmonary cone and pulmonary arteries. In contrast to cor pulmonale resulting from obstructive emphysema, in pulmonary embolism the enlargement of the inflow tract is usually more pronounced and, with it, the transverse diameter of the heart larger. The diaphragm is generally more elevated and the Valsalva effect on the heart which is so marked in obstructive emphysema is missing.

The elongation of the outflow tract of the right ventricle and the prominence of the pulmonary cone can become considerable. The apex of the heart is raised and, on palpation, the forceful positive epigastric thrust contrasts to the systolic depression of the apex beat.

The clinical findings characteristic of right heart failure are common to all forms of cor pulmonale. A correlation between questionable roentgenograms and electrocardiograms is always advisable. If, on lateral and oblique roentgenograms, there is a suggestive bulge of the right ventricle and the electrocardiogram shows evidence of right heart strain, as expressed by a depressed S-T and inverted T-waves, the diagnosis of cor pulmonale might well be considered. The history of previous phlebitis, evidence of an old infarction, absence or prominence of one of the pulmonary arteries, and exclusion of a pulmonary fibrosis, pneumoconiosis' and 'obstructive emphysema may suggest thromboembolism of the lung as a possible cause of cor pulmonale. Heart failure does not immediately follow.

Experimental investigations have shown that the right ventricle of the dog is able to compensate for compression of the pulmonary artery up to approximately 58 per cent of the total caliber, being able to overcome the added resistance, and to supply the left ventricle with a sufficient amount of blood. With a greater degree of compression of the pulmonary artery, however, the blood transfer to the left ventricle is diminished, the coronary blood supply decreased, and complete heart failure results.

It has been postulated that the mechanical effect of the pulmonary embolus is enhanced by the presence of vagal reflexes, which cause a widespread constriction of the pulmonary arterioles, a stimulation of the respiratory center, and have considerable influence upon the coronary blood flow.

Many of the arrythmias occurring with pulmonary embolism, tachycardia and tachypnea have been attributed to such reflexes arising within the involved lung and being transmitted through the cervical vasosympathetic nerves.

The frequency with which thrombophlebitis and migratory thrombi occur in malignancies has not been thoroughly investigated with the exception of linking them to the general debility and depletion of the patient. Thrombophlebitis is not infrequently found as the first significant sign of carcinoma of the pancreas and occurs, as well, in malignancy of the colon and in bronchogenic carcinoma.

Sproul found venous thrombosis in 32 out of 147 cases of cancer of the colon and in 12 out of 82 cases of cancer of the lung.

In Zimmerman and co-workers'[26] series of 56 patients who had pulmonary embolism, no less than 20 also had some form of malignancy. The tendency of sarcomas to grow upon and into blood vessels and their capacity to grow into the lumen without attachment to the vessel wall makes them

Fig. 5. Case IV. A forty-three year old female operated on twenty-one months previously for carcinoma of the ovary (total hysterectomy). Sudden complaints of severe chest pain and great weakness. Abrupt cut-off of the right pulmonary artery, transparent lower lung field. Large thrombus of the main pulmonary artery found at autopsy, but microscopic examination revealed tumor tissue, probably myogenic sarcoma, arising from arterial wall.

particularly prone to embolization and vascular obstruction.

CASE IV of our group is that of a forty-three year old female with a history of carcinoma of the ovary. A total hysterectomy, including removal of both ovaries, was performed in February, 1950. She was re-admitted twenty-one months later with complaints of severe chest pain radiating to the back and great weakness.

A chest roentgenogram taken on admission (Fig. 5) showed a prominent and abruptly terminating right pulmonary artery with a suggestion of a mass blocking or surrounding the pulmonary artery. The continuation of the vascular pattern consisted only of a few fine vascular lines and the lung was transparent in this area in spite of a small pleuritic fluid in the right costrophrenic angle. No metastases were discernible.

The patient died twenty-two days after admission. Autopsy revealed a large thrombus occluding both main branches of the pulmonary artery. Microscopic examination showed this thrombus to be formed by a tumor of sarcomatous nature. The tumor was very cellular, being composed of cells varying in size and shape, with bluish cytoplasm and central round nuclei. There were no metastases in the lung. The possibility of a myogenic sarcoma arising from the arterial wall or metastases from a hidden primary myogenic sarcoma was considered by the pathologist.

Comment. Roentgenologically, Case IV was indistinguishable from the following case, which illustrates the combination of bronchial asthma, cor pulmonale, polycythemia and large occluding emboli of the pulmonary artery.

CASE V was a fifty-four year old male who had been admitted to the hospital several times between 1944 and 1951 because of asthmatic attacks. He was afflicted with asthma since childhood and in later years developed a status asthmaticus. A severe polycythemia appeared which was treated with P^{32}. At the last admission, in December, 1954, he suffered from severe dyspnea and developed cerebral symptoms. He expired in January, 1955. A roentgenogram of the chest taken three weeks before death (Fig. 6) showed considerable enlargement of the right auricle, the right ventricle and, possibly, the left ventricle. A peculiar prominence of the right pulmonary artery with an abrupt termination was noted. The lungs appeared un-

FIG. 6. Case V. A fifty-four year old male with history of asthmatic attacks and polycythemia. Enlargement of the right heart, emphysema and right basal infarct. Note prominence and abrupt cut-off of right pulmonary artery. Great transparency of both lungs except in infarct area. Autopsy revealed occlusion of the pulmonary artery leading to the right lower lobe, right atrial appendage filled with thrombi (see text) and cor pulmonale.

expectedly clear with the exception of an infarct shadow and pleurisy in the right lung base.

On autopsy, the pulmonary artery leading to the right lower lobe was found occluded by a grayish-brown embolus. Proximal to the embolus, the right pulmonary artery was filled by a partially organized thrombus. The left pulmonary artery was partially occluded by an organized embolus which completely obstructed the branch of the left lower lobe. A likely source of the emboli was discovered in the right atrial appendage filled with thrombi in various stages of organization. Severe hypertrophy of the right ventricle was noted.

PULMONARY EMBOLISM LEADING TO INFARCTION

Only about one-half of all clinically recognized pulmonary emboli lead to infarction. Kjellberg and Olsen have presented experimental evidence in animals showing that as long as the bronchial arteries remain intact, no infarction is produced during artificial embolization. Pulmonary infarction is much more common in patients presenting a history of left ventricular heart failure, or mitral valve lesions with pulmonary congestion. The most frequent locations are the lower lobes, particularly in the periphery. The appearance of the infarct is so variable that many observers feel that a definite diagnosis is difficult. Yet, Hampton and Castleman[9] have, in their classic study on postmortem inflated and dissected lungs, shown the relationship of infarcts to lung segments and were able to establish a somewhat recurrent pattern.

One of these is the minimal pleuritic reaction of the costophrenic angle with a "hump-shaped" medial border. Careful observation will disclose the cut edge of the triangular or cuboid infarct resembling ice cubes in a glass of water. We have repeatedly found this sign very helpful in diagnosis (Fig. 7, *A* and *B*). The "wedge-shaped" appearance of the larger pulmonary infarct is more correctly described as a truncated cone, as the apex of the wedge directed towards the heart is blunted and cut off, leaving a clear zone of lung tissue between the peripheral density and the heart or

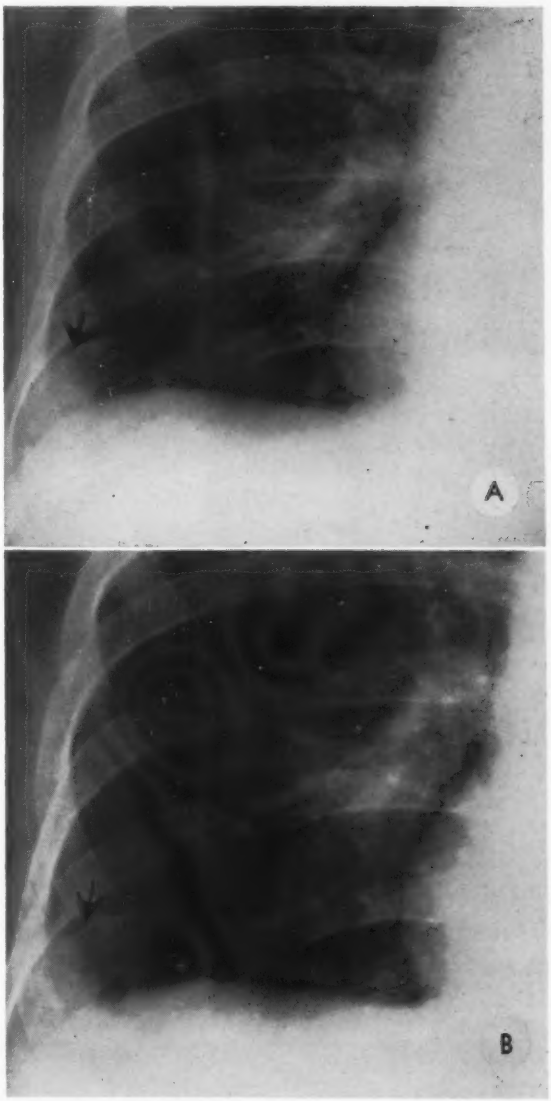

FIG. 7. (*A* and *B*) The characteristic basal angle infarct, its "hump-shaped ice-cube" appearance, with little fluid.

hilar shadow (Fig. 8). The initial thin-veiled shadow, which might easily escape detection becomes denser with time and might assume a round tumor-dense character. Upon rotation, however, and in various views, the round shadow can usually be made out as a "parachute" type of shadow with notched edges and a broad base resting on a pleural surface. As a general rule, infarcts always involve the adjacent pleural surface, which sometimes might be the interlobar fissure. They do not cause any

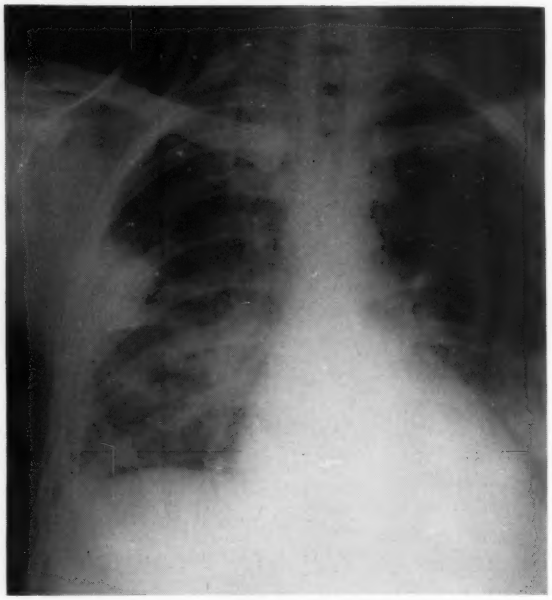

FIG. 8. The "truncated cone" appearance of a larger infarct. An incomplete, blunted wedge.

bulge such as a growth does. The pleurisy is usually localized and small insofar as it is caused by the infarct; it might contain bloody fluid. At times fibrinous pleurisy has been found instead (Fig. 9, *A* and *B*).

Infarcts occur in hidden locations and frequently assume the shape of a small meniscus or nugget of ambiguous character. We have found infarcts located in the parasternal region, in the posterior paramediastinal region, and in the upper lobes. In some instances a tenting of the diaphragm with a superimposed small triangular shadow was found to be an infarct. The smaller meniscus or cuboid-shaped infarct and the larger parachute, or, if we prefer, "parabolic" shadow, which usually terminates before it reaches the hilus, seemed to be the most constant diagnostic features (Fig. 10). On consecutive roentgenograms the density increases, while the extent decreases due to organization. They usually remain for a period of weeks and months, notwithstanding the fact that some "incomplete infarcts" might disappear within two to three days, leaving no visible scar. Others are known to leave scars, which assume the character of Fleischner's plate-like atelectasis and are usually found in the lung bases. Hampton and Castleman[9] found on dissection that their length is usually 1–4 cm. and that they reach the pleural surface and cause some dimpling of the pleura. The bronchioles in the surrounding area are rather wide and there is a compensatory emphysema. Since they represent actual scar tissue, we would, in contrast to Kerly lines, expect them to be single and rather firm. When they can be recognized, they are an important sign in a

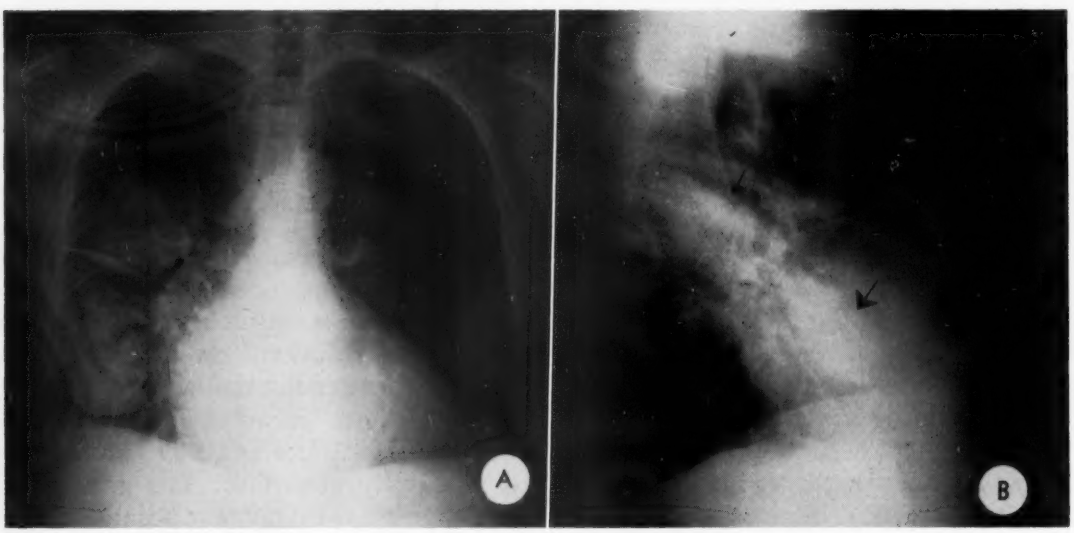

FIG. 9. (*A*) The parabolic or parachute type of consolidated infarct, which seems to hang in air but on lateral view (*B*) is firmly based on the lung interlobar fissure line.

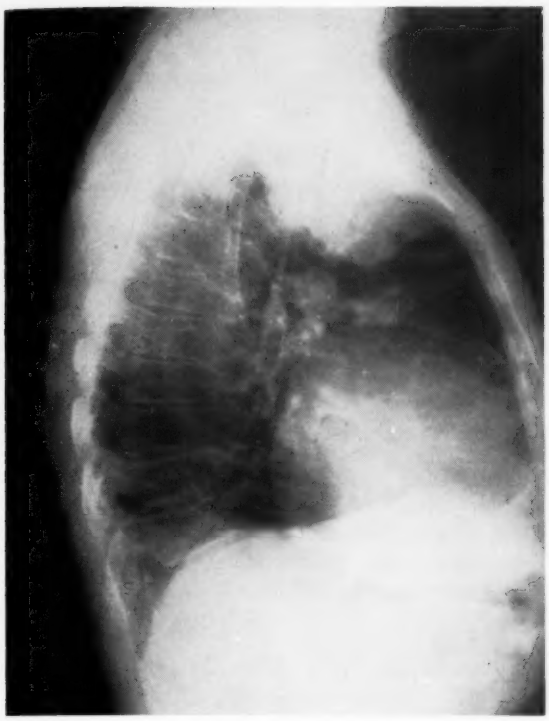

Fig. 10. Upper lobe infarct of parabolic type based on anterior pleural surface with notched irregular outline. Smaller "meniscus-like" infarct beneath it, above cardiac shadow.

condition which has a tendency to recurrence.

Considering the ever-increasing use of anticoagulants, enzymes and, most recently, molds with thrombus dissolving properties, it should frequently be possible for the radiologist with the cooperation of the clinician to make a more definite statement about pulmonary thromboembolism and infarction. Angiocardiography can be helpful in proving the presence and location of the vascular occlusion. The bedside roentgenogram is usually not satisfactory, and the patient should be brought, while in bed, into the radiologic department. A curved cassette or flexible industrial screen has many advantages; more than one view of the chest is necessary. None of the roentgenologic or clinical signs are infallible or always present, but awareness of the frequency and circumstances of occurrence of thromboembolism in the lungs is a prerequisite of the proper diagnosis.

SUMMARY

1. The incidence of thromboembolism of the lung of either surgical or medical origin has remained high in spite of greater clinical awareness and better methods of prevention, vein ligation and anticoagulant treatment. The clinical signs and symptoms are frequently suggestive. Roentgenography or angiocardiography is in a high percentage of cases decisive. Thromboembolism without infarction presents as an abnormal transparency of the involved lung segment (Westermark) with absence of one or both of the large hilar vessels. The heart shadow assumes a peculiar shape, best described as the "amphoric" type. In the absence of both hilar vessels (pulmonary arteries), a large saddle embolus must be considered.

A thrombus or embolus which is located slightly more peripherally within the main branches of the pulmonary arteries causes a buckling and aneurysmal widening of the vessel, with either a sharp cut-off or tapering at the peripheral end of the embolus. Such thrombus or embolus is compatible with life, and leads to the development of either acute or chronic cor pulmonale. In contrast to the cor pulmonale of obstructive emphysema, the position of the diaphragm is elevated, the heart does not assume the vertical position and develops within a short time enlargement of inflow and outflow tracts of the right ventricle.

2. Thromboembolism with infarction does not, as a rule, present Westermark's sign of increased translucency of a lung segment, since it occurs mostly in a congested lung with dilated bronchial arteries and numerous collaterals. Infarction is common in mitral stenosis, hypertensive heart failure and coronary insufficiency. Infarcts represent an area of ischemic necrosis usually located at the periphery of the lung. They are rarely "wedge-shaped," as the apex of the wedge is cut off. A "truncated cone" or an "umbrella" shape results which is quite characteristic. Smaller infarcts frequently have a cuboid or meniscus-

like appearance. In the costophrenic angle the "hump-shaped" elevation on the medial side of a small pleurisy has frequently been observed and appears diagnostic. The base of the embolic infarcts is sometimes the interlobar fissure line, along which they form a parabolic shadow, which is best seen on a lateral roentgenogram.

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THE IMPORTANCE OF CHEST ROENTGENOGRAPHY IN THE DIAGNOSIS OF PULMONARY EMBOLISM*

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EVIDENCE of pulmonary embolism is reported in 10 to 25 per cent of necropsies.[10,14] Although in many instances embolism is an incidental finding or is only a contributory factor toward death, the clinical recognition of minor episodes of embolism is of practical importance since effective treatment is available which offers a means of preventing fatal massive embolism.

Unfortunately, in many hospitals and in office practice, pulmonary embolism frequently goes unrecognized. Clinically, embolic episodes are commonly misdiagnosed as pleurisy, pneumonia, and coronary artery disease. There are no pathognomonic symptoms and signs to indicate the correct diagnosis, and no specific laboratory tests which may be used to establish the presence of thromboembolism. Electrocardiographic changes reflecting right heart dilatation due to acute pulmonary hypertension are frequently demonstrable if serial examinations are made; but the abnormalities are often misinterpreted as indicative of primary myocardial disease.

The importance of roentgenologic examination in the diagnosis of pulmonary disease and the key role which the radiologist has assumed in this field need little emphasis. It is evident from experience in many hospitals that the frequency with which pulmonary embolism is clinically diagnosed depends largely on the frequency with which the radiologist calls attention to the possibility of this disease. A number of excellent reports in recent years have been concerned with the roentgenologic diagnosis of pulmonary embolism and infarction. Hampton and Castleman,[3] Krause and Chester,[7] Miller and Berry,[8] and Short[12] have emphasized that roentgen changes following embolism show no specifically diagnostic characteristics, and that in some instances no roentgen changes are demonstrable. Unfortunately, the radiologist may not only fail to call attention to the possibility of embolism; but he may dispel a clinical suspicion of pulmonary embolism by reporting changes which he regards as indicative of pneumonia or pleurisy, or by reporting that the lungs are normal in appearance.

Short,[12] in a clinical study in England, observed pulmonary embolism to be the most common acute pulmonary disease encountered in modern hospital practice. Our experience at the Graduate Hospital in recent years has accorded with Short's observations; embolism is now diagnosed more often than pneumonia or bronchogenic carcinoma.[4]

MATERIAL

The diagnosis of pulmonary embolism was made on the basis of clinical, laboratory or necropsy findings in 90 patients at the Graduate Hospital from August 1, 1955 to January 31, 1957. The 90 cases represented an attack rate of 0.09 per cent among 10,130 adults admitted during this interval. The rate among admissions to the medical service was 1.2 per cent and among those admitted to surgical services 0.6 per cent. Thirty-three patients had embolism following surgery. An equal number entered the hospital as the result of pulmonary embolism. Of the 90 patients with embolism, 69 recovered from their illness, and 21 died within the period of the study.

* From the Departments of Radiology and Medicine, Graduate Hospital of the University of Pennsylvania. Presented at the Fifty-ninth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., September 30–October 3, 1958.

† Teaching and Research Fellow of the American Trudeau Society.

Nine of the deaths were due to pulmonary embolism; in the other fatalities, pulmonary embolism was merely contributory or incidental. Analysis of the clinical findings indicated that embolism produced predominantly pulmonary manifestations in 43 per cent of the patients, cardiac manifestations in 37 per cent and abdominal syndromes in 7 per cent. Neurologic and miscellaneous symptoms occurred in 13 per cent. Clinical evidence of phlebitis was found in only 61 per cent of patients but the lower extremities were believed to have been responsible in all but a minority whose emboli were attributed to sources in the arms, heart and abdomen. Significant electrocardiographic changes were observed in 71 per cent of the 75 patients who had serial tracings. The high frequency of transient electrocardiographic abnormalities is especially remarkable since the episodes of embolism were often of moderate or slight severity.

Roentgenographic examination of the chest was secured following the onset of symptoms attributable to embolism in 72 of the patients. Roentgenograms were not secured in 18 instances either because the embolism was quickly fatal or because the diagnosis of pulmonary embolism was not suspected until necropsy was performed. The present study represents an analysis of the 72 cases, with special emphasis on assessing the value of roentgenographic examination in the diagnosis of pulmonary embolism. The roentgenograms have been reviewed and compared with the original reports of these studies. The roentgen characteristics and distribution of lesions have been tabulated. Serial films, available in most surviving patients, provided an opportunity to measure the rapidity of clearing of the roentgen abnormalities.

INCIDENCE OF ROENTGEN ABNORMALITIES

Abnormal densities were observed in the roentgenograms of 60 patients. In 12 instances, the roentgenographic examination of the chest was interpreted as showing no abnormality. Subsequent review of these roentgenograms with the knowledge that a diagnosis of pulmonary embolism had been established by other methods produced no change from the original interpretation. The percentage with normal chest roentgenograms in the present study (16.6 per cent) is slightly higher than the 9.5 per cent reported by Short,[12] but is slightly lower than the 22 per cent noted by Miller and Berry.[8]

INTERVAL BETWEEN ONSET OF SYMPTOMS AND ROENTGEN CHANGES

Of the 27 patients examined within twenty-four hours of the onset of symptoms, 5 had normal roentgenograms. Twenty-three patients were examined twenty-four to forty-eight hours after the onset of symptoms; 4 had normal chest roentgenograms (Table 1). Only one of the 9 patients with negative chest roentgenograms subsequently showed evidence of infarction. Short[12] and Hampton and Castleman[3] have pointed out that it may be several days after embolism before roentgen changes appear, but our experience suggests that the delayed appearance of roentgenographic densities is relatively uncommon.

ROENTGEN CHANGES FOLLOWING EMBOLISM

Almost any configuration of parenchymal pulmonary abnormality may fol-

TABLE I

TIME ELAPSED BETWEEN ONSET OF SYMPTOMS AND INITIAL ROENTGEN EXAMINATION OF THE CHEST FOLLOWING A PULMONARY EMBOLISM

Graduate Hospital 1955–1957

Time Elapsed	No. with Abnormal Findings	No. with Normal Findings
Less than 24 hr.	22	5
1 to 2 da.	19	4
2 to 4 da.	7	2
Over 4 da.	12	1
Total	60	12

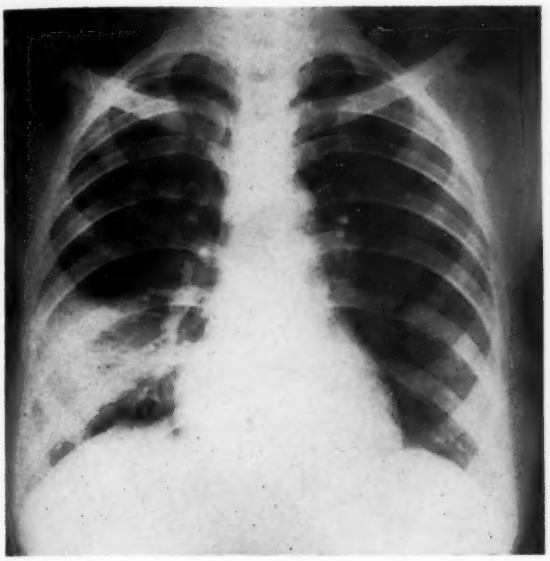

Fig. 1. Chest roentgenogram of a thirty-nine year old female with bilateral thrombophlebitis of the legs, cough and right sided chest pain made forty-eight hours after onset of symptoms. There is a homogeneous density at the right base which completely resolved after anticoagulant therapy.

low embolism. The most common changes which we noted, however, were either a moderate-sized homogeneous density (Fig. 1) or linear densities adjacent to a pleural surface and associated with an effusion (Table II).

Table II

ABNORMAL ROENTGEN CONFIGURATIONS FOLLOWING PULMONARY EMBOLISM

Graduate Hospital 1955–1957

Abnormality	No. of Cases
Pleural effusion	33*
Homogeneous densities	27
Elevation of diaphragm	21
Linear densities	19
Patchy densities	12
Cavitation	1

* Seven large effusions, 26 small effusions.

A sharply demarcated homogeneous density was observed in 27 patients; linear densities were noted in 19 patients. A pleural effusion (Fig. 2, *A* and *B*) was present in 33 cases, and a pleural surface was involved by the embolic process in 45 patients.

The diaphragm was abnormally elevated on the affected side in 21 patients. In 16 cases there were associated pleuropulmonary changes; in 5 instances the elevation of the diaphragm was the only roentgenographic abnormality. Necrosis and cavitation within an infarct were observed in one instance. No patient in this series demonstrated an ischemic lung following

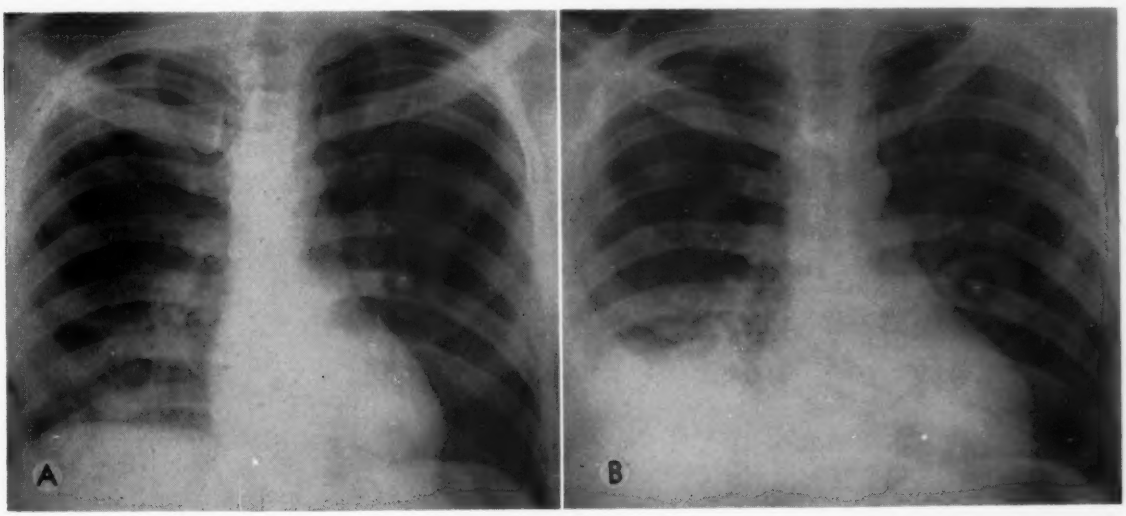

Fig. 2. (*A*) December 5, 1957: Roentgenogram showing normal chest made four hours after onset of right sided pleuritic pain. (*B*) December 7, 1957: Roentgenogram made two days later shows a homogeneous density and pleural effusion at the right base. Intercostal pulmonary biopsy performed because of suspicion of tuberculosis revealed an organized infarct.

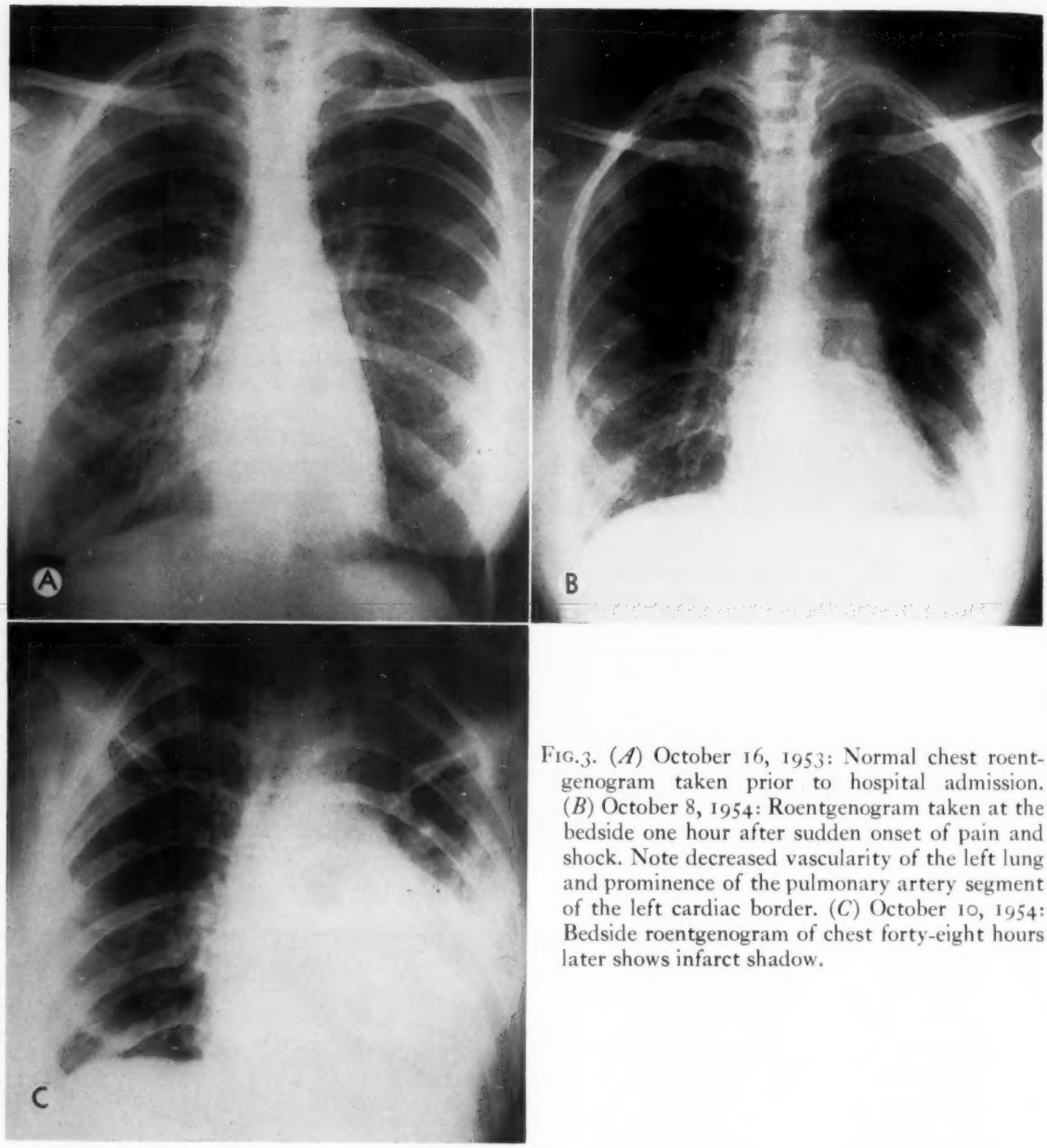

FIG. 3. (*A*) October 16, 1953: Normal chest roentgenogram taken prior to hospital admission. (*B*) October 8, 1954: Roentgenogram taken at the bedside one hour after sudden onset of pain and shock. Note decreased vascularity of the left lung and prominence of the pulmonary artery segment of the left cardiac border. (*C*) October 10, 1954: Bedside roentgenogram of chest forty-eight hours later shows infarct shadow.

embolism although all roentgenograms were reviewed with this possibility in mind; nor did any patient exhibit prominence of the pulmonary conus sector of the cardiac silhouette. In a patient seen in this hospital in 1954, however, both of these changes were shown (Fig. 3, *A*, *B* and *C*). It would appear that the ischemic lung, as described by Westermark[16] and by Shapiro and Rigler,[11] is a relative rarity.

The dimensions of the lesions demonstrated on roentgenograms correspond closely with those observed by Hampton and Castleman.[3] In the present series, 52 per cent measured more than 4 cm. in diameter, 38 per cent measured between 2 and 4 cm. in diameter and 10 per cent measured from 1 to 2 cm. in diameter. None was recognized which measured less than 1 cm. in diameter.

Table III

ROENTGEN CHANGES AFTER PULMONARY EMBOLISM

Graduate Hospital 1955–1957

Anatomic Distribution	
Right upper lobe	5
Right middle lobe	2
Right lower lobe	21
Left upper lobe	4
Left lower lobe	14
Right lower lobe, Left lower lobe	12
Right upper lobe, Left lower lobe	1
Right middle lobe, Right lower lobe	1
	60

DISTRIBUTION OF THE LESIONS

Forty-six of the 60 patients with abnormal chest roentgenograms had only one area of involvement. The lower lobes were affected most frequently (76 per cent of cases); in 21 instances on the right side and in 14 on the left (Table III). This corresponds with the distribution noted by others.[5,8,13] The upper lobe was the site of embolism in 9 patients, in 5 on the right and in 4 on the left side. Two patients had changes involving the right middle lobe.

Fourteen patients had two lobes involved. Twelve of these had changes in both lower lobes; one had abnormal findings in the right upper lobe and left lower lobe, the other had involvement of the right middle and lower lobes.

DURATION OF ROENTGEN CHANGES

Although it is generally considered that the roentgen changes due to pulmonary embolism are of brief duration, in only one of our cases did the lesion completely resolve in less than three days (Table IV). Four patients exhibited complete or almost complete resolution of abnormal shadows within one week, 6 within two weeks and another 6 within the next two weeks. In 19 or 52 per cent of patients with adequate follow-up, definite changes were still present more than four weeks after the initial abnormal chest roentgenogram. Similar observations were reported by Short.[12]

Table IV

DURATION OF ROENTGEN CHANGES AFTER ONSET OF SYMPTOMS OF PULMONARY EMBOLISM

Graduate Hospital 1955–1957

Less than 8 da.	5
8 to 14 da.	6
15 to 28 da.	6
More than 28 da.	19
Incomplete follow-up	24

CORRELATION OF ORIGINAL CLINICAL DIAGNOSIS WITH ORIGINAL ROENTGENOGRAPHIC INTERPRETATION

Comparison of the original roentgenographic interpretation with the clinical diagnosis recorded on the request for roentgenologic consultation indicates that a clinical diagnosis of pulmonary embolism was made in 43 instances. Of these, 7 had normal chest roentgenograms, and 7 had abnormalities noted by the radiologist which were *not* reported as pulmonary infarctions. Subsequent review by the authors showed the roentgen findings in the latter 7 to be compatible with pulmonary infarction. The remaining 29 patients with a clinical diagnosis of pulmonary embolism exhibited changes which the radiologist regarded as due to pulmonary embolism.

The original roentgenographic request reflected no clinical suspicion of pulmonary embolism in 29 cases. In 10 instances the radiologist was able to suggest this diagnosis on the basis of the roentgen findings. The diagnosis of embolism was considered neither by the clinician nor the radiologist at the time of the initial examinations in the remaining 19 cases.

The roentgen diagnosis of pulmonary infarction was suggested by the radiologic interpreter in a total of 39 cases (Table V). Pulmonary infarction was the only roentgen diagnosis offered in 21 patients. Eigh-

TABLE V

INITIAL ROENTGENOLOGIC DIAGNOSES IN 72 PATIENTS EXAMINED AFTER PULMONARY EMBOLISM

Graduate Hospital 1955–1957

	No. of Patients
Negative chest	12
Pneumonitis	9
Atelectasis	3
Pleurisy with effusion	2
Congestion	2
Others	5
Infarction or pneumonitis	12
Infarction or atelectasis, congestion, etc.	6
Infarction only diagnosis suggested	21

teen reports suggested pulmonary infarction or some other possibility: pneumonitis was mentioned 12 times; atelectasis, pleural effusion, pleural thickening and localized emphysema were each suggested as a possible diagnosis once; pulmonary congestion was suggested as a possibility in 2 patients.

In the remaining 21 cases showing roentgenographic abnormalities, the report made no mention of pulmonary infarct. Eight other etiologic possibilities were suggested: pneumonitis, pleural effusion, atelectasis, congestion, tuberculosis, metastasis, pleurisy, and subdiaphragmatic abscess.

COMMENT

As a result of widespread misconceptions regarding the clinical and roentgenographic manifestations of pulmonary embolism, in many hospitals this diagnosis is rarely made except in fatal cases. A striking increase in the frequency of diagnosis of pulmonary embolism in recent years at the Graduate Hospital has resulted from the collaboration of radiologists with other staff physicians especially interested in the disease, who have utilized all clinical, electrocardiographic and other laboratory aids in an effort to detect minor episodes of embolism.

A common cause for the failure of the radiologist to suggest the diagnosis is his lack of familiarity with the varied changes in the chest roentgenogram which follow pulmonary embolism. The old concept[15] that the roentgen diagnosis can be made only when one encounters a "wedge-shaped density with its apex directed toward the hilum" is hard to dispel even though the variable manifestations of pulmonary infarction have been described frequently.[5–8,12,13,16]

Hampton and Castleman,[3] in their fundamental study of pulmonary embolism, observed that embolism without infarction usually results in a relatively normal chest roentgenogram. When infarction occurs, nothing abnormal may be seen during the first twelve hours. At the end of twenty-four hours, changes are usually evident, but these are not sharply defined and may resemble roentgenologically the changes noted in bronchopneumonia. Later the area of involvement becomes sharply defined. They emphasize, however, that the configuration noted roentgenographically depends on the site of involvement, and that the true extent of the pathologic process can be appreciated only when roentgenograms are exposed in various projections. Thus an infarct in the base of the lung near the costophrenic sulcus in the posteroanterior projection may simulate so-called pleural thickening. An infarct involving part of the middle lobe may mimic an interlobar effusion or atelectasis when seen in only the posteroanterior view (Fig. 4).

Since patients with pulmonary embolism are frequently too ill to have multiple exposures made in various degrees of rotation, the roentgenologic study must often be restricted to a single anteroposterior exposure made at the bedside with portable apparatus. It is, therefore, important that the radiologist familiarize himself with the more common variations of a pulmonary infarct as seen in this projection.

A patchy density or a well-circumscribed homogeneous density similar to those described by Hampton and Castleman[3] was

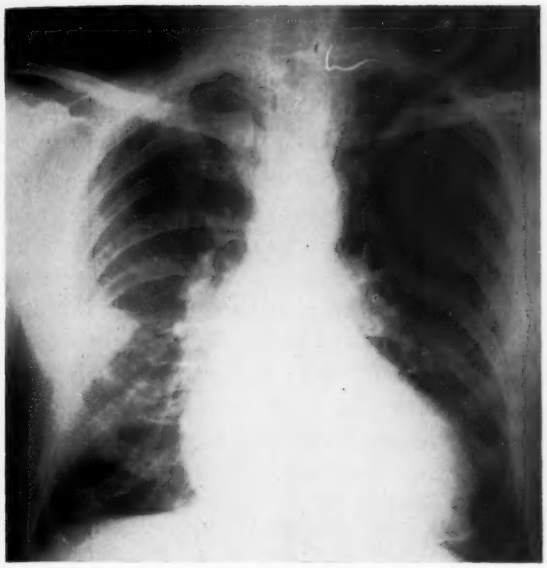

Fig. 4. Chest roentgenogram of a fifty-seven year old male with known hypertension. The patient developed hemoptysis and dyspnea followed by chills and fever. Roentgenogram reveals well circumscribed density in the lateral portion of the right mid lung field. After treatment with anticoagulants and diuretics, the density resolved slowly over a period of three weeks, indicating that the changes were not due to cardiac decompensation only.

Fig. 5. Roentgenogram of the chest of a fifty-six year old female with evidence of thrombophlebitis of the left leg showing density in the left mid lung field. She was treated with anticoagulants for ten days with no change in the size of the lesion. Periodic re-examinations for a period of six weeks showed no change. Lobectomy performed because of suspicion of neoplasm disclosed an organized infarct.

the most common lesion observed in our patients (Fig. 5), but a significant number (21.6 per cent) showed linear densities. Similar observations were reported by Short.[12] Possible causes for the linear densities including those described by Fleischner and his co-workers[2] are as follows: (1) Not seeing the fullest extent of the infarct in a single view; (2) interlobar pleuritis with or without effusion; (3) areas of focal atelectasis; and (4) scarring of the infarct when the roentgenograms are made late in the process.

In our experience, the typical pattern of roentgen changes in the chest after embolism is as follows: Moderate-sized homogeneous densities are observed in one or both lower lobes. Extension to a pleural surface is usually demonstrable, and a small pleural effusion is commonly present. Linear densities resembling plate-like atelectasis are seen in the same or contralateral lung. Infarct shadows may resolve rapidly, but more often clear gradually with development of one or more linear scars. In many instances, the original roentgenographic study may be compatible with pneumonia, and only when serial roentgenograms show characteristic changes will pulmonary embolism be suggested roentgenologically.

Occasionally a pulmonary infarct may be so well circumscribed that primary or metastatic neoplasm is simulated (Fig. 5). Perkins and Bradshaw[9] have reported several instances in which pulmonary resections performed for suspected tumor revealed only organized infarct.

Although effusion is a common finding following pulmonary embolism, the effusion is usually small and of little clinical importance. Attention should be called, however, to the not infrequent instances in which pulmonary embolism is associated with a large effusion. These cases require differentiation from tuberculous pleurisy with effusion (Fig. 6).

The roentgen diagnosis of infarct was suggested in 39 patients in this series. Twenty-one patients had definitely abnormal findings described in the original

FIG. 6. Chest roentgenogram of a fourteen year old male showing large right pleural effusion. The patient had right sided chest pain and a high fever. The tuberculin test was negative. There was a history of leg injury ten days prior to admission. A phlebitis of the right leg was noted on examination. The symptoms subsided after institution of anticoagulant therapy and the pleural effusion gradually disappeared.

report but misinterpreted most commonly (12 cases) as pneumonitis. Upon review of these roentgenograms for the purpose of the present report, the changes in all were considered to be consistent with pulmonary infarction. Thus the important fact is not that the diagnosis was suggested in slightly more than half of the cases, but that there were changes present in more than 80 per cent of the examinations which should lead to the correct interpretation by radiologists who are familiar with the roentgenologic and clinical manifestations of this common and important disorder.

SUMMARY AND CONCLUSIONS

1. The roentgenographic changes observed in 72 patients following pulmonary embolism have been studied. Abnormalities were noted in 60 instances. Pleural effusions (33 instances) and homogeneous densities resembling those produced by pneumonia (27 instances) were the most common findings. Elevation of the diaphragm was noted in 21 patients, usually in association with some parenchymal abnormality. Linear densities, presumably representing focal areas of atelectasis or small interlobar effusions, were observed in 19 patients.

2. Densities appeared rapidly following embolism. Abnormal findings were observed as frequently in roentgenograms made within twenty-four hours of the onset of symptoms as in those made subsequently. Densities resulting from embolism were slow to resolve. Abnormal findings cleared within a week in 5 cases and persisted for more than four weeks in 19 cases.

3. The correct diagnosis was suggested by the radiologist in 10 cases in which the possibility of pulmonary embolism had not been clinically considered. The diagnosis of embolism was suggested by the radiologist in only 54 per cent of the entire series. The radiologist's failure to consider this diagnosis in the other cases must be attributed to misconceptions concerning the roentgenologic criteria for diagnosis of pulmonary embolism and lack of familiarity with the clinical manifestations of this disease.

4. The alert radiologist will consider pulmonary embolism in the differential diagnosis of every acute cardiorespiratory illness. He will suggest this as a possibility in all patients with pulmonary and pleural densities resembling pneumonia and pleurisy. Not infrequently, he will recognize characteristic roentgenographic patterns that will provide strong evidence for a diagnosis of pulmonary embolism even when typical clinical manifestations may be absent.

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PULMONARY MANIFESTATIONS IN COLLAGEN DISEASES*

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THE term "collagen disease" was originally applied by Klemperer and his colleagues[21] in 1941 to a group of diseases of obscure etiology distinguished by a systemic alteration of the connective tissue, especially of its extracellular components, throughout the body. Histologically, this group is characterized by the accumulation of fibrinoid,[22] a homogeneous, eosinophilic refractile material which has some of the tinctorial properties of fibrin.

Collagen diseases include polyarteritis nodosa, disseminated lupus erythematosus, scleroderma, dermatomyositis, rheumatic fever and rheumatoid arthritis.[11,14] These conditions may overlap, clinically and pathologically, and rheumatoid arthritis rather frequently occurs as part of the clinical picture of the others. Some authors have also included Schönlein's purpura, thrombotic thrombocytopenic purpura, erythema nodosum, some cases of glomerulonephritis, serum sickness, transfusion reactions, thromboangiitis obliterans and ulcerative colitis in the collagen group.[33,37]

Another controversial disease not seen in this series but sometimes considered as a collagen disease is Wegener's granulomatosis.[38] This is a syndrome characterized clinically by sinusitis, necrotizing granuloma of the upper and lower portions of the respiratory tract, renal insufficiency, arthralgia and diffuse vasculitis with a circumscribed and occasionally cavitating pulmonary parenchymal lesion which may resemble metastatic disease. This may be a renal-respiratory subtype of polyarteritis nodosa.

Although the etiology of the collagen diseases remains obscure, much clinical and experimental evidence indicates that the development of a state of hypersensitivity plays an impotant role.[3,7,10,31] Historical, clinical, laboratory and pathologic data are reviewed in the extant medical literature.[8,32,33] This presentation is concerned chiefly with roentgen manifestations in the thorax. Other pertinent roentgenologic data will be included when indicated to be of diagnostic value.

MATERIAL AND METHODS

For this study the case records, pathologic data and roentgenograms were reviewed of 109 patients admitted to the University of Minnesota Hospitals from 1942 to 1956 inclusive. Five conditions, namely, polyarteritis nodosa, disseminated lupus erythematosus, scleroderma, dermatomyositis and rheumatic pneumonitis, are included. Controversial conditions have not been included, and rheumatoid arthritis does not usually present thoracic manifestations except when it occurs in conjunction with these diseases or when far advanced cases have thoracic cage involvement which leads to nonspecific inflammatory changes. The incidence and means of diagnosis of these five diseases in this series are given in Table 1.

Those patients who were diagnosed by means of autopsy or biopsy were considered as proved cases. Those patients who were diagnosed by means of the clinical history, physical examination and laboratory tests were considered as clinical diag-

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TABLE I

INCIDENCE AND MEANS OF DIAGNOSIS IN 109 PATIENTS WITH FIVE COLLAGEN DISEASES

Disease	Means of Diagnosis		Clinical	Total
	Autopsy	Biopsy		
Polyarteritis nodosa	17	9	14	40
Disseminated lupus erythematosus	8	21	8	37
Scleroderma	4	8	8	20
Dermatomyositis	2	3	2	7
Rheumatic pneumonitis	3	0	2	5
Totals	34	41	34	109

noses only. In the course of our study, tables were made in which laboratory data and roentgen signs were tabulated in "proved" and "clinical" groups. Since the data did not differ appreciably, the tables were combined for further reference.

ROENTGEN FINDINGS

Significant pathologic lesions were found in the lungs, pleura or heart in 28 out of 34 autopsies in this series. Since roentgen signs were demonstrated in these organs in approximately two-thirds of the entire series of 109 patients, including those still living, it seems reasonable to assume that if all patients were examined periodically until death, roentgen signs would appear in the chest during some phase of the disease in over 80 per cent of all patients with these five diseases.

Interstitial pneumonitis, pleural effusion and a nonspecific type of cardiac enlargement occurred with sufficient frequency in all five diseases[25,37] so that these findings may be considered as confirmatory in nature but are not usually of great aid in the differential diagnosis of these conditions. Some degree of atelectasis also may occur in all of them, although it was notably lacking in our patients with scleroderma.

There are several other roentgen signs in the thorax which occur in one or more of the collagen diseases, but which are rather nonspecific when considered alone. For example, hilar vascular prominence occurs in several clinical conditions, but, in the collagen group, it is most often seen in polyarteritis nodosa.[23,32,35] Interstitial pulmonary fibrosis and emphysema are also seen in many clinical conditions, but in the collagen group they are probably most common in scleroderma and dermatomyositis.[5,24,28,30] A honeycomb appearance or visualization of multiple small cyst-like shadows in the lower two-thirds of the lungs is rather characteristic of scleroderma, and when this sign is present it is definitely of diagnostic value.[6,9,15,16]

Pericardial effusion or pericarditis was demonstrated in some of the patients of all groups except dermatomyositis, but was found more often in disseminated lupus erythematosus and rheumatic pneumonitis in this series.[26,29] Both of these tend to involve the endocardium and multiple serous surfaces so that valvular lesions, pericardial and pleural involvements are common to both.[1,2,19]

Small nodular lesions are found in the pulmonary parenchyma in polyarteritis nodosa and scleroderma. Since polyarteritis tends to involve small arteries, it is not surprising that small areas of consolidation appear in the lungs occasionally. They suggest the appearance of small infarcts.[12,35,36]

Two patients with polyarteritis suffered a unilateral spontaneous pneumothorax and one patient with dermatomyositis developed bronchiectasis.

Roentgen signs are demonstrated less frequently in other organs, but some may on occasion be of confirmatory and even of differential diagnostic value. Dilatation and lack of peristalsis in the esophagus, absorption of terminal phalanges, and calcification of soft tissues, especially in weight-bearing areas, are all helpful signs in the diagnosis of scleroderma.[4,17] Absorption of the peridontal membrane has also been reported in some patients,[34] but the presence of this sign was not studied in this series.

Most of these conditions can involve the small and large intestines on occasion to produce localized areas of intestinal distention.[14,20] This occurs most frequently in polyarteritis and scleroderma among the collagen group. Of special interest in this regard are 2 patients in this series who developed polyarteritis distal to the resected segment of coarctation of the aorta. The only significant roentgen finding in these patients who complained of abdominal pain after surgery was a moderate adynamic ileus.

Roentgen evidences of rheumatoid arthritis were found in 3 patients with polyarteritis, 3 with scleroderma and 2 with dermatomyositis.

In this series it was noted that many roentgenographic signs regressed following clinical improvement with steroid therapy. This regression can be quite striking in patients with polyarteritis and disseminated lupus erythematosus.[25]

The incidence of the various roentgen signs occurring in this series of 109 patients is given in Table II. From even a cursory glance at this table it will be noted that most of the signs appear to be relatively nonspecific or of confirmatory nature, especially when considered separately. However, various combinations of signs or sequential changes occurring in the thorax may suggest the presence of a collagen disease in roughly one-third of the patients some time in the course of the disease.

REPORT OF CASES

The cases reported here are selected from this series with a view to emphasizing roentgen signs which may occur in the thorax in collagen diseases and illustrating how combinations of these signs may be analyzed to be of diagnostic significance.

CASE 1. K.B., a male aged forty, was admitted May 24, 1950. The complaints were

TABLE II

INCIDENCE OF ROENTGEN FINDINGS IN COLLAGEN DISEASES

	Polyarteritis Nodosa	Disseminated Lupus Erythematosus	Scleroderma	Dermatomyositis	Rheumatic Pneumonitis
No. of Patients	40	37	20	7	5
A. Pulmonary Changes					
Hilar enlargement	14	0	0	0	0
Pneumonitis	13	17	9	5	5
Pulmonary edema	8	c	0	1	3
Nodular lesions	5	c	3	0	0
Pleural effusion	5	12	8	2	1
Atelectasis	4	9	0	1	1
Pulmonary fibrosis	0	3	7	3	0
Emphysema	0	5	7	0	0
Honeycomb or cystic appearance	0	0	4	0	0
Pneumothorax	2	0	0	0	0
Infarction	1	0	c	0	0
Bronchiectasis	0	0	0	1	0
B. Other Changes					
Cardiac enlargement	14	7	9	3	5
Pericarditis	1	6	0	0	3
Ileus	5	1	2	0	0
Rheumatoid arthritis	3	0	3	2	0
Aperistaltic esophagus	0	0	4	0	0
Calcification in soft tissues	0	0	2	1	0
Absorption of phalanges	0	0	1	0	0

arthritis, convulsions, fever, headache, and loss of weight of ten to twelve weeks. Physical examination: duplicated mitral second sound, blood pressure 180/130. Urine: two plus albumin, red blood cells, granular and red blood cell casts. Hb 13.7 gm., white blood cells 19,600, 88 per cent neutrophils. Erythrocyte sedimentation rate 73 mm./hour. Serum protein 7.1 gm., albumin 2.6 gm., globulin 4.5 gm. Electrocardiogram: left axis deviation.

Roentgen examination: May 25, 1950: heart normal, hilar shadows prominent. May 31, 1950: heart enlarged, hilar shadows prominent, bilateral pulmonary edema. June 7, 1950: perihilar, pulmonary edema, progressive dilatation of heart. July 12, 1950: heart larger, prominent hilar vascular shadows and bilateral pneumonitis; also perihilar edema (developed after renal failure). July 15, 1950: right pneumothorax (Fig. 1, *a* and *b*).

Autopsy August 6, 1950: polyarteritis nodosa, fibrinous pericarditis, myocardial fibrosis, coronary arterial sclerosis, small infarcts in lungs and kidneys, and congestion of liver.

Comment. The combination of pneumonitis, pleural effusion, nonspecific cardiac enlargement and hilar vascular prominence in the absence of peripheral pulmonary vascular engorgement suggested the presence of widespread changes in small vessels rather than cardiac or renal failure. This combination is not always present, but, when it is seen, it is strongly in favor of polyarteritis nodosa. Pulmonary edema usually appears after renal failure, and hypertension usually indicates that some of the renal lesions are in a healing phase. The urinary sediment indicates that both acute and chronic changes are present in the kidneys. This has been called telescopic urinary sediment.

Spontaneous pneumothorax is uncommon in patients with collagen disease and occurred only in polyarteritis in this series. Possibly, it is related to pulmonary infarction with subsequent rupture of alveolar walls or blebs.

Case II. P., a female aged eighty, was admitted October 13, 1953. Her history revealed intermittent muscle pains and fever for eight years, retroverted uterus, and vaginal bleeding for which she had received injections of estrogens. Blood pressure 176/?. Urine: two plus albumin. Hb 11.7 gm., white blood cells 9,650. Erythrocyte sedimentation rate 84 mm./hour. Reversal of A-G ratio. Electrocardiogram: slightly prolonged R-S interval.

Roentgen examination: October 13, 1953: isolated nodule in right lung, slightly enlarged left ventricle, perivascular markings prominent. December 1, 1953: pulmonary nodule has

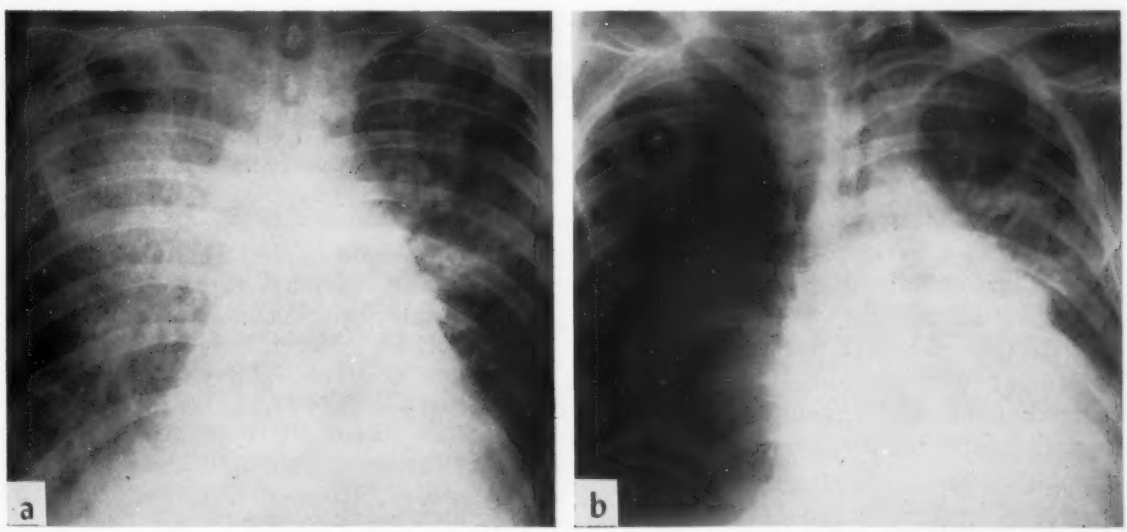

Fig. 1. Case 1. Polyarteritis nodosa. (*a*) July 12, 1950: cardiac enlargement, prominent hilar shadows, and bilateral pneumonitis. Perihilar edema developed after renal failure. (*b*) July 15, 1950: spontaneous pneumothorax, right side (pulmonary infarct at autopsy August 6, 1950).

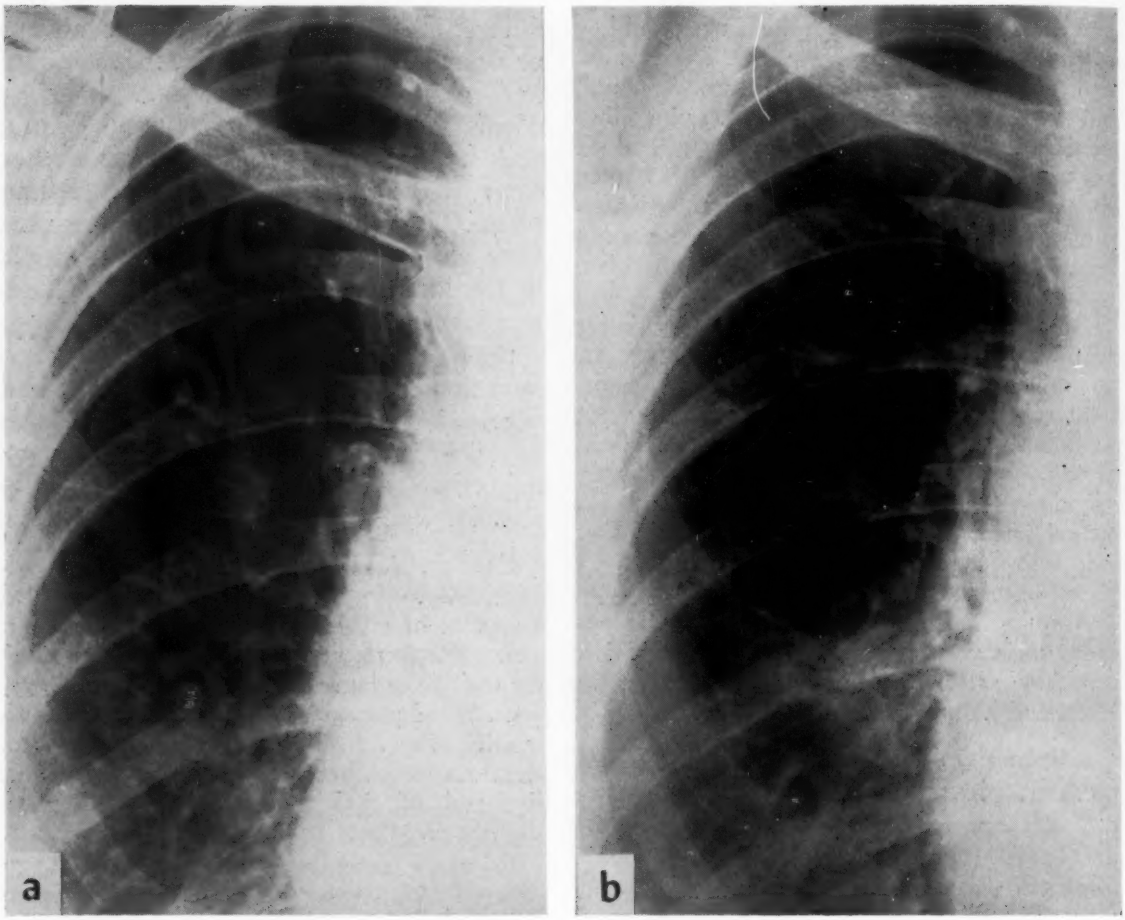

FIG. 2. Case II. Polyarteritis nodosa. (*a*) October 13, 1953: isolated nodule in right lung, slightly enlarged left ventricle, and prominent perivascular markings. (*b*) December 1, 1953: pulmonary nodule has disappeared during cortisone therapy.

disappeared during cortisone therapy (Fig. 2, *a* and *b*).

Comment. The presence of slight left ventricular enlargement and peripheral pulmonary vascular changes might lead one to suspect that the isolated pulmonary nodule might be secondary to cardiac disease. Clinical evidence of a systemic disease and the disappearance of the nodule during treatment with cortisone indicated that the pulmonary nodule could well have been a small infarct produced by local change in a small vessel.

CASE III. R.R., a male aged fifty-seven, was admitted February 21, 1952. His complaints were paroxysmal nocturnal dyspnea for one year, asthmatic attacks, hemoptysis, chills, abdominal pain and petechial rashes on legs. White blood cells 8,900, 21 per cent eosinophils. Erythrocyte sedimentation rate 102 mm./hour, reversed A-G ratio. Lymph node biopsy: compatible with polyarteritis nodosa. Good response to ACTH and cortisone, eosinophilia disappeared.

Roentgen examination: February 13, 1952: chest negative. March 8, 1952: prominent hilar shadows, interstitial inflammatory changes with small nodules (Fig. 3 *a*). March 28, 1952: small nodules cleared, hilar shadows less prominent (Fig. 3 *b*). June 25, 1952: signs of bronchopneumonia. August 26, 1952: lungs clear.

The patient was discharged on April 12, 1952 as apparently cured following ACTH and cortisone therapy. Later he suffered an episode of bronchopneumonia which also cleared.

Comment. The enlarged hilar shadows and nodular interstitial pneumonitis, es-

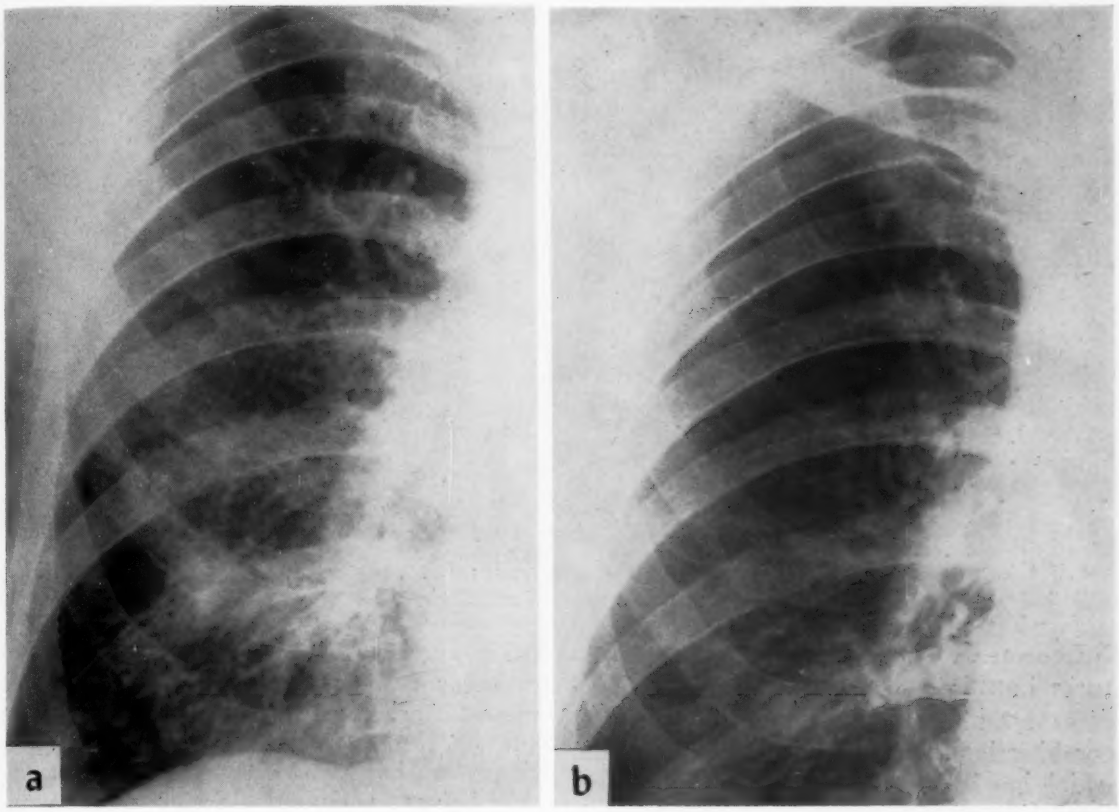

FIG. 3. Case III. Polyarteritis nodosa. (*a*) March 8, 1952: prominent hilar shadows, interstitial pneumonitis with small nodules. (*b*) March 28, 1952: small nodules cleared, hilar shadows less prominent following steroid therapy.

pecially in the absence of cardiac enlargement and peripheral pulmonary vascular engorgement, were strongly suggestive of polyarteritis nodosa. The regression of these changes following steroid therapy offered further confirmation.

CASE IV. A.M., a female aged thirty-nine was admitted on February 18, 1951, with complaints of fever, cough, emaciation and diarrhea. No skin lesions on admission but LE cell test positive. Later a typical butterfly skin rash appeared on the face. Crepitant rales in both pulmonary bases and a Grade II apical systolic murmur were noted. Urine: one plus albumin, with red and white blood cells. Erythrocyte sedimentation rate 86 mm./hour. White blood cells 4,350. Reversal of A-G ratio. Electrocardiogram: myocardial damage.

Progress: The patient improved under cortisone and prednisone therapy. She was discharged, but when steroids were continued she developed hallucinations and other mental symptoms. She was admitted to the hospital again and treated with chlorpromazine. Later she developed respiratory and cardiac symptoms and pericardial effusion. Recurrence and regression were noted many times until death on March 21, 1956 (over five years after original hospital admission).

Roentgen examination: February 28, 1951: interlobar effusion and pneumatocele in right middle lobe (Fig. 4, *a* and *b*). August 16, 1951: pneumatocele smaller. June 17, 1952: atelectasis and pneumonitis in right lung, pleural thickening, cardiac silhouette enlarged. January 25, 1954: atelectasing pneumonitis involving most of right lung, cardiac silhouette larger. March 12, 1956: pneumonitis of right lung, cardiac silhouette considerably enlarged, suggesting pericardial effusion.

Autopsy March 21, 1956: disseminated lupus erythematosus. Pulmonary vessels showed thickening of vessel walls and intimal proliferation. Pulmonary atelectasis, edema, and bronchopneumonia; peribronchial fibrosis. Adhesive pleuritis and pericarditis. Wire-looping of renal glomeruli. Atrophy of adrenals.

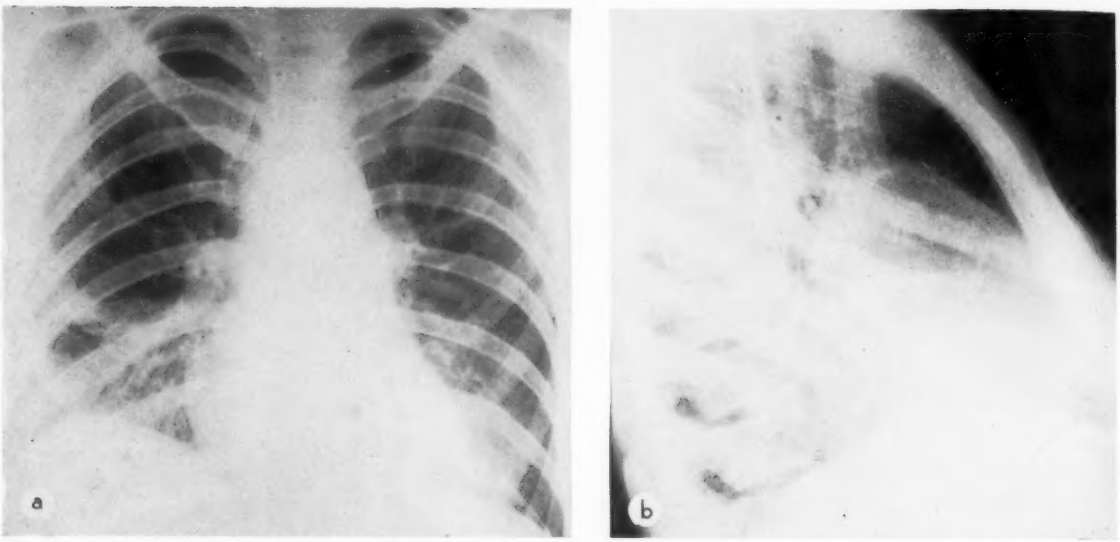

FIG. 4. Case IV. Disseminated lupus erythematosus. (*a*) February 28, 1951: pneumatocele right middle lobe, pneumonitis. (*b*) Lateral view.

Comment. The distinctive roentgen features in this case were polyserositis with pleural and pericardial effusion and the combination of pneumonitis and atelectasis which has been termed "atelectasing pneumonitis." These were characteristic, though perhaps not pathognomonic, of disseminated lupus erythematosus, and together with other clinical and laboratory data constituted a striking clinical picture.

CASE V. E.H., a female aged forty was admitted February 26, 1953 with complaints of chronic fatigue for four years, chills, fever and multiple skin rashes, with butterfly distribution on nose and cheeks and also on exposed areas of chest and extremities. Physical examination: Temperature 102° F., heart enlarged, rhythm irregular, apical systolic murmur. Urine: one plus albumin, two plus red blood cells and granular casts. White blood cells 3,300. Hb 3.8 gm. Erythrocyte sedimentation rate 104 mm./hour. Reversed A-G ratio. Electrocardiogram: right axis deviation. LE cell test positive in peripheral blood and bone marrow.

Roentgen examination: February 27, 1953: lungs clear, cardiac silhouette enlarged. December 30, 1953: atelectasis and pneumonitis in right upper lobe. Appearance of cardiac silhouette indicated pericardial effusion (Fig. 5).

Autopsy January 1, 1954: disseminated lupus erythematosus, with typical wire-looping of renal glomerular capillaries, bilateral bronchopneumonia, pulmonary emphysema, bilateral pleural effusion, pericardial effusion, peritoneal effusion, chronic lymphedema of lower extremities, hemorrhagic necrosis and fibrosis of spleen, skin lesions consistent with lupus erythematosus.

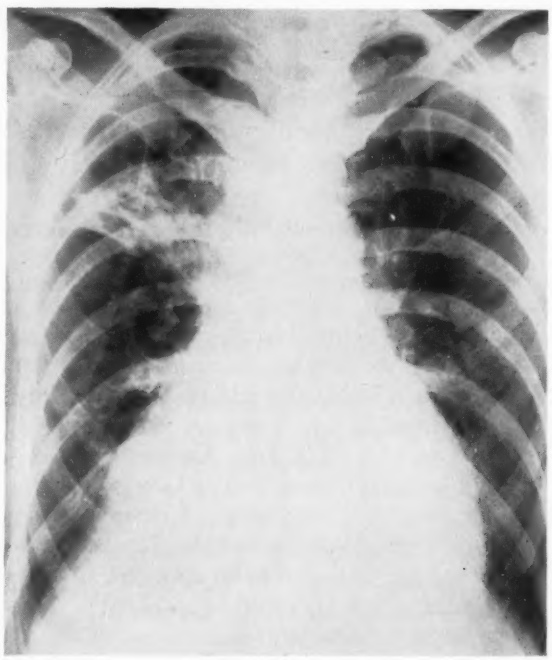

FIG. 5. Case V. Disseminated lupus erythematosus. Atelectasis and pneumonitis in right upper lobe, enlarged cardiac silhouette probably due to pericardial effusion.

Comment. The clinical and laboratory data in this patient were diagnostic. The autopsy findings of polyserositis, renal and splenic lesions were equally diagnostic. The findings of atelectasis, pneumonitis and pericardial effusion occurred rather late as a combination of signs, but were strongly confirmatory, and, if this combination were seen earlier in the course, it would have aided in the differential diagnosis.

CASE VI. S.G., a female aged forty-seven was admitted February 22, 1954 with complaints of Raynaud's phenomenon for several years, myalgia, arthralgia migrating from upper to lower extremities, residual paresis of left side of face from previous hemiparesis, flexion deformity of fingers, and thickening of skin over forehead, arms and fingers. Physical examination: Thickened skin, flexion of fingers, split second sound and gallop rhythm at apex of heart, scattered rales in lungs. Urine: albumin and hyaline casts. Reversed A-G ratio. Electrocardiogram: left axis deviation, left ventricular strain and interventricular conduction defect. Blood clot test negative for LE cells.

Roentgen examination: July 22, 1953: interstitial pneumonitis and fibrosis, with multiple small cyst-like areas in lungs, generalized cardiac enlargement, encapsulated pleural effusion on right, free fluid on left. March 6, 1954: pneumonia with bilateral basal pleural effusion, heart larger. Also encapsulated pleural effusion on right (Fig. 6).

Autopsy October 12, 1954: generalized scleroderma of skin, also involving esophagus; pulmonary fibrosis and edema; cardiac hypertrophy; myocardial fibrosis; fibrinous pericarditis and effusion.

Comment. The combination of interstitial pneumonitis and fibrosis, with multiple small cyst-like areas usually occurring in the middle and lower thirds of the lungs especially when accompanied by nonspecific cardiac enlargement and pleural effusion in the absence of peripheral pulmonary vascular engorgement, should be strongly suggestive of scleroderma. An esophagram showing dilatation and lack of peristalsis in the lower thirds of the esophagus would have been of further confirmatory value in this patient.

FIG. 6. Case VI. Scleroderma. Interstitial pneumonitis and fibrosis with multiple small cyst-like areas in lungs, generalized cardiac enlargement. Encapsulated effusion on right side.

CASE VII. M.E., a female aged fifty-two, was admitted March 23, 1955 with complaints of Raynaud's phenomenon for twenty-five years, increasing dyspnea for fifteen years, gradually developing skin pigmentation and dysphagia. The patient had had a coronary attack in 1951. A skin biopsy at Mayo Clinic disclosed scleroderma. Physical examination: heart enlarged, systolic murmur Grade III loudest at left fourth intercostal space; blood pressure 110/20; basal pulmonary rales; subcutaneous calcification right upper arm. Urine: trace of albumin. Reversed A-G ratio. Electrocardiogram: right axis deviation, right ventricular strain and first degree heart block.

Roentgen examination: December 15, 1949: interstitial fibrosis, emphysematous and cyst-like changes in base of right lung. March 24, 1955: Calcification of subcutaneous soft tissue (Fig. 7 *a*). Esophagram: widening and lack of peristalsis (Fig. 7 *b*). More calcification in soft tissues. April 7, 1955: slight increase in pulmonary fibrosis and pneumonitis; cyst-like changes in base of right lung; enlarged heart (Fig. 7 *c*).

Progress: discharged on April 22, 1955 as improved after treatment with digitoxin, aminophyllin and multivitamins.

Comment. The presence of interstitial

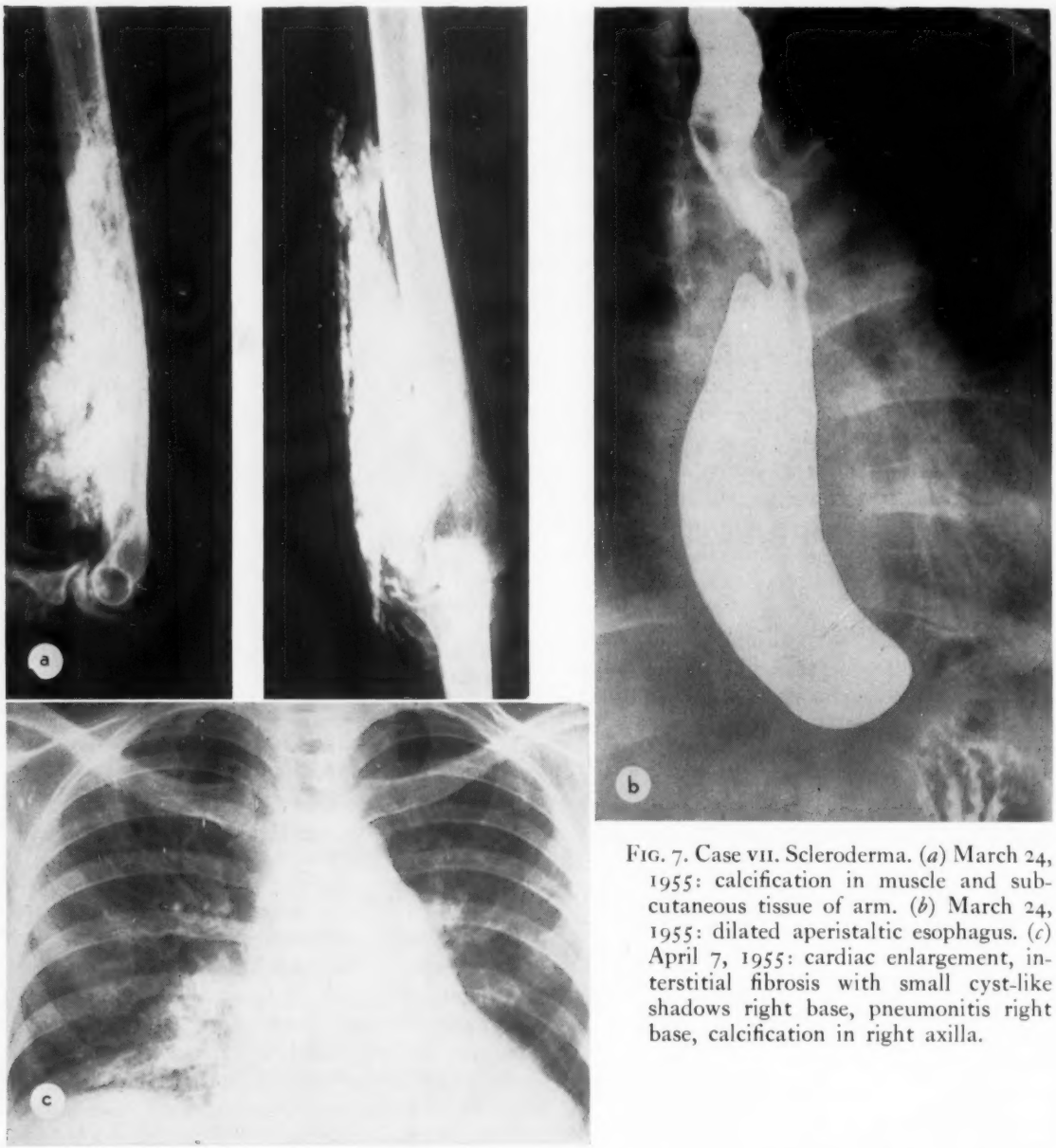

FIG. 7. Case VII. Scleroderma. (*a*) March 24, 1955: calcification in muscle and subcutaneous tissue of arm. (*b*) March 24, 1955: dilated aperistaltic esophagus. (*c*) April 7, 1955: cardiac enlargement, interstitial fibrosis with small cyst-like shadows right base, pneumonitis right base, calcification in right axilla.

fibrosis, emphysematous and cyst-like changes in the basal pulmonary tissue was suggestive. The combination of these with nonspecific cardiac enlargement, lack of peristalsis and widening of the esophagus and subcutaneous calcification should be considered diagnostic of scleroderma. A well developed case of dermatomyositis might also show all of these findings, but none of our cases showed the peculiar cyst-like pulmonary changes.

CASE VIII. S., a male aged fifty-one, was admitted on February 19, 1953 with complaints of Raynaud's phenomenon, fever, cough, and a tight and thick skin over the forehead. He had been investigated at Glen Lake Sanatorium for tuberculosis and discharged as nontuberculous. Skin biopsy revealed scleroderma.

Roentgen examination: extensive fibrosis and ring-like shadows in both upper lobes; bilateral basal emphysema; heart normal; pleural thickening left (Fig. 8).

Progress: symptomatic relief with cortisone therapy. Discharged as improved on March 6, 1953.

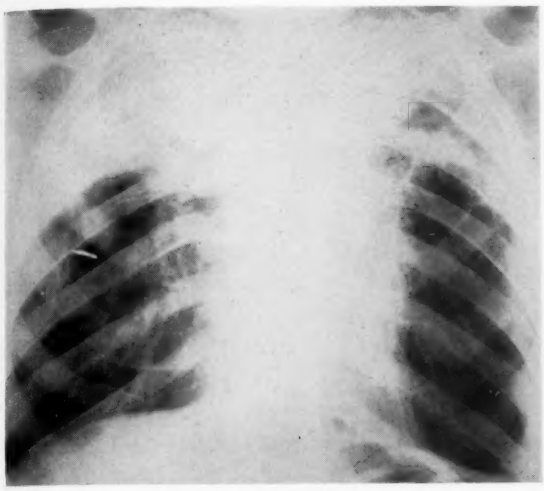

Fig. 8. Case viii. Scleroderma. Unusual location of dense interstitial fibrosis and multiple small cyst-like shadows in upper lobes. Bilateral basal emphysema. Pleural thickening of left base.

Comment. This was an exceptional case of scleroderma in that the fibrosis and ring-like shadows occurred in both upper lobes and led to a suspicion of pulmonary tuberculosis. The interstitial fibrosis and peculiar ring-like shadows are actually much more characteristic of scleroderma. The presence of basal emphysema and pleural thickening, when combined with the above and especially when no tubercle bacilli can be demonstrated by appropriate tests, should be strongly in favor of scleroderma. The addition of the clinical finding of tight, rigid skin over the forehead should make the clinical diagnosis secure.

Case ix. M.J., a female aged twenty-one was admitted December 11, 1949 with complaints of pain and weakness in leg muscles and joint pains for six weeks, rash on face and body, and fever. Physical examination: temperature 101.2° F., blood pressure 95/60, soft systolic murmur over precordium. Urine negative. Erthyrocyte sedimentation rate 102 mm./hour. Reversed A-G ratio. Electrocardiogram: low amplitude. Muscle biopsy: myositis, compatible with diagnosis of dermatomyositis.

Roentgen examination: December 13, 1949: chest negative. March 28, 1950: cardiac silhouette enlarged. March 25, 1950: tuberculous infiltration of left upper lobe. June 7, 1950: left pleural effusion; rheumatoid arthritic changes in knees. July 11, 1950: no pleural fluid; tuberculous infiltration of left upper lobe stationary (Fig. 9 *a*). September 1, 1950: Subcutaneous calicification of both arms (Fig. 9 *b*).

Progress: Improved on physiotherapy and discharged. Walked independently when seen in July, 1955.

Comment. This case was complicated by pulmonary tuberculosis. The presence of nonspecific cardiac enlargement combined with calcification of the soft tissues in the arms was strongly suggestive of a collagen disease, especially dermatomyositis. The demonstration of rheumatoid arthritic changes in the knees offered further confirmation of a collagen disease. The differentiation from scleroderma required biopsy.

Case x. R.L., a male aged seven and one-half, was admitted February 9, 1955 with complaints of a red rash on nose, anorexia, irritability, low grade fever, pain in the legs, easy fatiguability and swelling in the periorbital areas. Physical examination: temperature 99.6° F., pulse 120. Hot, dry patches on the skin. Palpable axillary and cervical lymph nodes. Grade ii systolic murmur along left sternal border. Second pulmonic sound split. Enlarged liver. Contraction and atrophy of the muscles of the extremities. Laboratory findings: positive urinary albumin, white blood cells 10,950, reversal of A-G ratio. No LE cells in blood.

Roentgen examination: February 11, 1955: chest negative. April 30, 1955: slight cardiac enlargement, minimal bilateral interstitial changes (Fig. 10 *a*). November 28, 1955: increased cardiac enlargement, bilateral interstitial pneumonitis. January 14, 1956: heart larger, increase in bilateral interstitial pneumonitis (Fig. 10 *b*). January 27, 1956: massive atelectasis of left lung, interstitial and bronchopneumonia in right lung (Fig. 10 *c*).

Progress: treatment with cortisone, metacortone and testosterone ineffective.

Autopsy January 27, 1956: massive atelectasis of left lung, interstitial and bronchopneumonia, fibrinoid deposits in alveolar walls. Dermatomyositis.

Comment. The gradual development of nonspecific cardiac enlargement and interstitial pneumonitis without signs of cardiac

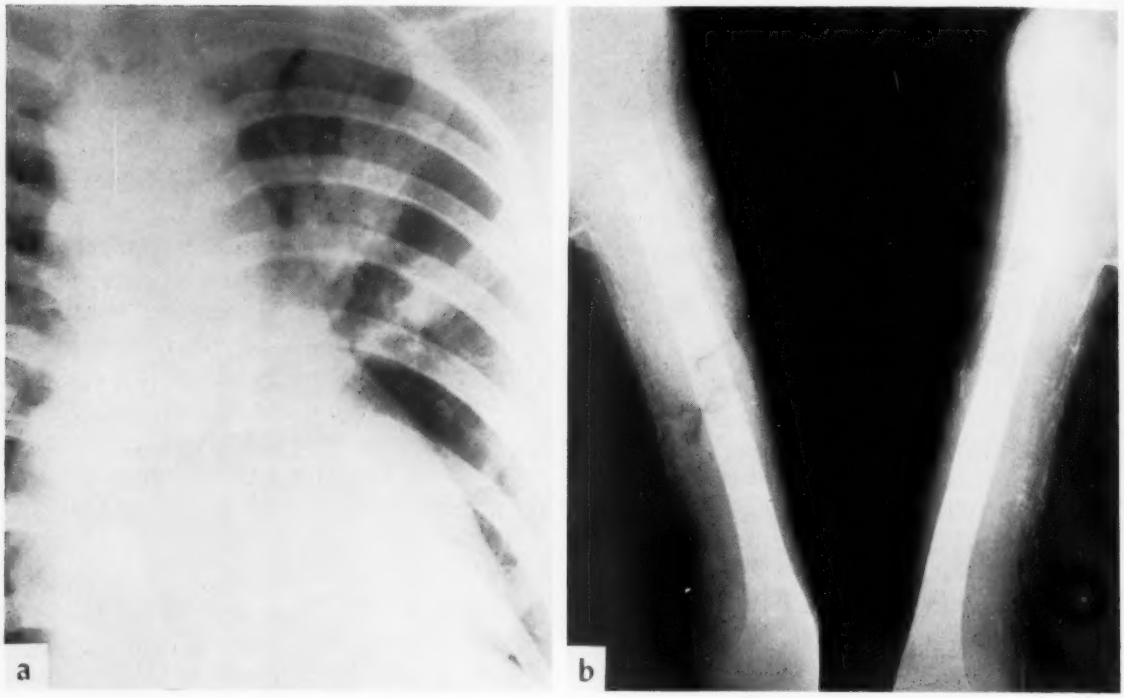

FIG. 9. Case IX. Dermatomyositis. (*a*) July 11, 1950: stable tuberculosis in left upper lobe, enlarged heart. (*b*) September 1, 1950: calcification of subcutaneous tissue of arms, atrophy of muscles.

failure indicated the possibility of a collagen disease and were strongly confirmatory when considered with other clinical and laboratory signs. The roentgen findings were not of further differential diagnostic value and could be seen in any of the collagen disorders. In a patient of this nature biopsy is necessary to establish the diagnosis of dermatomyositis. The terminal massive atelectasis of the left lung is unusual and not of definite diagnostic aid.

CASE XI. L.S., a female aged thirty-nine was admitted June 6, 1955 with complaints of general debilitation, dysphagia, weakness and soreness of muscles of upper extremities, skin rash and arthralgia. Physical examination: temperature 98.4° F., pulse 92, blood pressure 140/80. Marked weakness and atrophy of shoulder and upper arm muscles, erythematous patchy skin lesions on the posterior aspect of neck, back and dorsum of hands. Laboratory findings: urine normal. Erythrocyte sedimentation rate 49 mm. per hour. LE cell test negative. Skin and muscle biopsy: dermatomyositis.

Roentgen examination: June 7, 1955: chest negative. Feburary 1, 1956: chest negative. February 12, 1956: bilateral interstitial pneumonitis. March 29, 1956: heart normal, increase in bilateral interstitial pneumonitis (Fig. 11). April 2, 1956: pneumonitis resolving. April 16, 1956: enlarged heart, bilateral basal bronchopneumonia, probable pulmonary edema.

Progress: In spite of treatment with cortisone and penicillin the patient developed a progressive anemia, cardiomegaly, thrombophlebitis and electrolytic imbalance. She expired on May 11, 1956. Autopsy refused.

Comment. The roentgen signs in the chest were developed relatively late and were largely confirmatory. The development of bilateral interstitial pneumonitis was not diagnostic, especially when it occurred prior to the development of cardiac enlargement. The progressive enemia and development of cardiac enlargement were accompanied by the onset of pulmonary edema and basal pneumonia which obscured the roentgen diagnostic picture.

CASE XII. O.R., a male aged nine, was admitted July 9, 1952 with complaints of sore

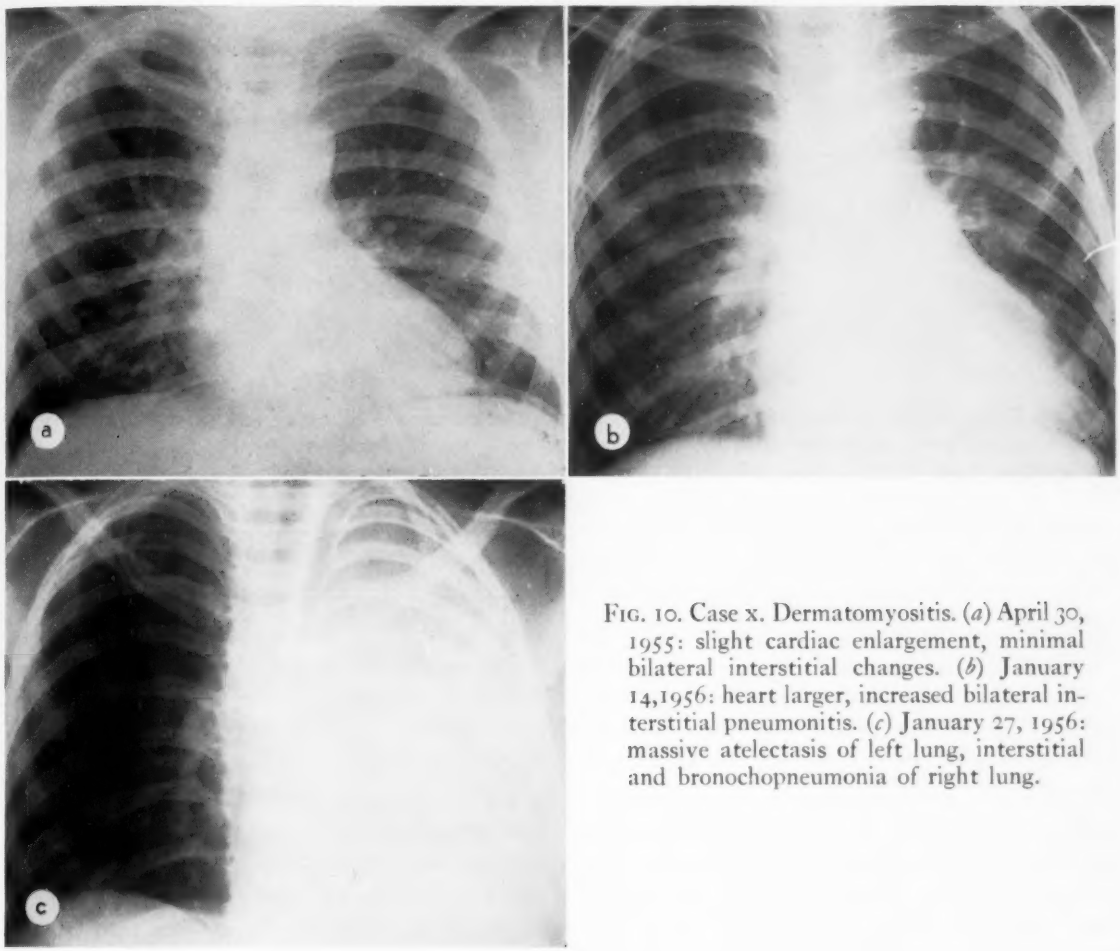

Fig. 10. Case x. Dermatomyositis. (*a*) April 30, 1955: slight cardiac enlargement, minimal bilateral interstitial changes. (*b*) January 14,1956: heart larger, increased bilateral interstitial pneumonitis. (*c*) January 27, 1956: massive atelectasis of left lung, interstitial and bronochopneumonia of right lung.

throat, high fever, increasing respiratory difficulties, weakness and joint pain. There was a history of glomerulonephritis in April, 1951. Physical examination: temperature 101.6° F., pulse 140, blood pressure 110/40. Heart enlarged to left. Apical systolic murmur, aortic diastolic murmur. Lungs clear at first, later bronchial breathing. Laboratory findings: urine negative. Erythrocyte sedimentation rate 90 mm./hour. White blood cells 23,450. Electrocardiogram: left ventricular preponderance and hypertrophy. Three plus C—reactive protein tests.

Roentgen examination: July 9, 1952: generalized cardiac enlargement, bilateral basal pneumonitis without peripheral pulmonary vascular engorgement (Fig. 12 *a*). July 24, 1952: pulmonary edema, vascular engorgement, abrupt cardiac enlargement probably due to pericardial effusion (Fig. 12 *b*).

Fig. 11. Case xi. Dermatomyositis. Bilateral interstitial pneumonitis. Heart normal.

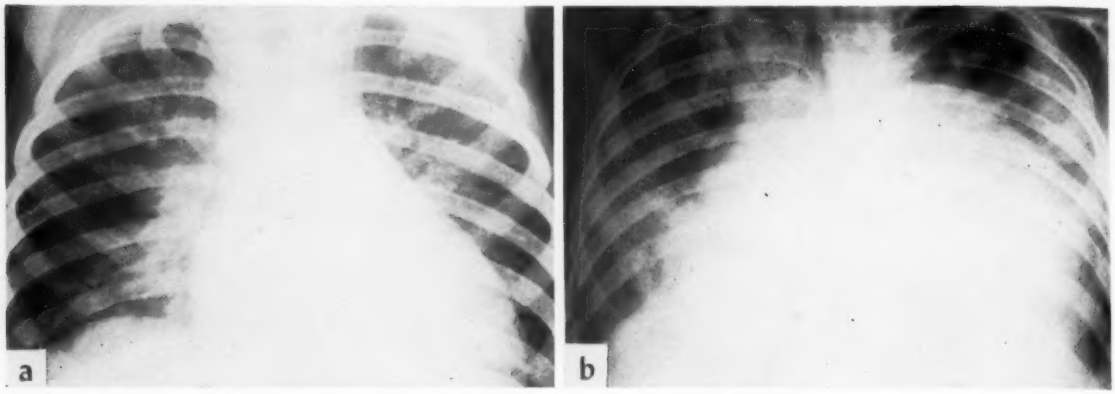

FIG. 12. Case XII. Rheumatic pneumonitis. (*a*) July 9, 1952: generalized dilatation of heart, bilateral pneumonitis and pleural effusion without peripheral pulmonary vascular engorgement. (*b*) July 24, 1952: abrupt increase in cardiac size (effusion), pulmonary vascular engorgement and edema.

Autopsy July 25, 1952: acute rheumatic fever; acute active myocarditis and valvulitis; fibrinous pericarditis; rheumatic pneumonitis with hyaline membrane around alveoli; old plastic peritonitis.

Comment. In a patient suspected of having rheumatic heart disease the roentgen demonstration of pneumonitis in the absence of peripheral pulmonary vascular engorgement and edema should lead one to consider rheumatic pneumonitis. Obviously, if fully developed cardiac failure were present when the patient was first seen, the demonstration of pneumonitis would be more difficult.

DISCUSSION

From a review of Table II and the case reports it is readily seen that the roentgen manifestations of collagen diseases in the chest present a complicated picture that is rather difficult to describe. Some of the patients have normal roentgenograms of the chest, especially in the earlier phases of the disease. Often other clinical and laboratory findings enable one to make a specific diagnosis before any roentgenographic changes are present.

In this series of 109 patients the roentgenograms of the chest probably did not contribute to the diagnosis at any time in about one-third of the patients. In another one-third, one or two abnormalities in the chest appeared at some phase of the disease process as confirmatory findings which could be considered of help in following the progress of the patient. Of special interest to the roentgenologist, however, is the final one-third of patients in which a combination or sequence of roentgen findings should actually lead one to suspect a collagen disorder. If there are clinical and laboratory findings suggesting the presence of a collagen disease these roentgenographic changes will aid in the differentiation of the various types of collagen diseases in about one-fifth of the patients.

It should be of interest to consider certain combinations or sequential roentgen changes which should cause one to suspect a collagen disease. The combination of interstitial pneumonitis and pleural effusion is somewhat suggestive, and the addition of prominent hilar shadows or nonspecific cardiac enlargement or effusion, especially in the absence of peripheral pulmonary vascular engorgement and edema, makes the presence of a collagen disease even more likely.

The combination of interstitial fibrosis and emphysema is not diagnostic. However, when these changes are more prominent in the lower two-thirds of the lungs, when there are small cyst-like shadows, when there is nonspecific cardiac enlargement or pleural effusion, and in the absence of signs of cardiac failure, a collagen disease

TABLE III

COMBINATIONS OF ROENTGEN SIGNS IN COLLAGEN DISEASES

	Thoracic Signs	Non-Thoracic Signs
Collagen diseases in general	Pneumonitis Pleural effusion Cardiac enlargement	Evidence of rheumatoid arthritis
Polyarteritis nodosa	Hilar vascular prominence Single or multiple nodules	Ileus, small bowel changes
Disseminated lupus erythematosus	Combination of atelectasis and pneumonitis Polyserous involvement	Ileus
Systemic scleroderma	Fibrosis Emphysema Cardiac enlargement Ring-like or cyst-like shadows or tiny nodulations	Aperistaltic esophagus Absorption of phalanges Soft tissue calcification Ileus, small bowel changes
Dermatomyositis	Fibrosis Emphysema Cardiac enlargement	Soft tissue calcification Aperistaltic esophagus and small bowel changes possible
Rheumatic pneumonitis	Cardiac enlargement and pneumonitis before or between episodes of failure	

such as scleroderma should be suspected.[6,30,39]

Nonspecific cardiac enlargement, pneumonitis and pleural effusion occur with sufficient frequency in all five of the diseases under discussion so that they are of little differential diagnostic value when viewed singly or in combination. When there is pericardial involvement, and especially when both pericardial effusion and pleural effusion are present, it would seem that the diagnosis of disseminated lupus erythematosus or rheumatic pneumonitis should be favored.[29]

The presence of hilar vascular prominence in combination with other findings mentioned in the paragraph above is strongly in favor of polyarteritis nodosa.[23,35] The presence of tiny pulmonary nodules, fleeting episodes of pneumonitis or pulmonary infarction may also be found in polyarteritis.[13] Spontaneous pneumothorax occurred only in 2 patients with polyarteritis. Adynamic ileus or partial intestinal obstruction also occurred more frequently in polyarteritis.

The combination of pleural and pericardial effusion or the presence of atelectasis and pneumonitis or combinations of these findings, are in favor of disseminated lupus erythematosus. The typical skin rash and the LE cell test are the strongest differential points clinically.[18,19,27]

Interstitial pulmonary fibrosis is common to both scleroderma and dermatomyositis. If multiple small cyst-like shadows are present scleroderma is more likely. A dilated aperistaltic esophagus is also seen in scleroderma. Small bowel or colonic involvement, leading to ileus or duodenal stasis, and absorption of the distal phalanges are uncommon signs but definitely helpful.[17] Widening of the peridontal space also has been given as a sign in scleroderma.[34]

In dermatomyositis the pulmonary fibro-

sis is not likely to present tiny nodulations and the peculiar multiple small cyst-like shadows so characteristic of scleroderma. In extensive cases with soft tissue calcifications, biopsy is necessary to differentiate dermatomyositis and scleroderma.

The combination of rheumatic cardiac enlargement and pneumonitis,[26,29] occurring before or between episodes of cardiac failure (vascular engorgement, pulmonary edema), forms the chief clue in detecting rheumatic pneumonitis. When signs of failure are present, namely, vascular engorgement and pulmonary edema, the pneumonitis may be obscured. Pulmonary edema is also seen with diffuse cardiac enlargement in the other collagen diseases, and of itself is rather nonspecific, usually indicating cardiac or renal failure.

The various combinations of roentgen signs are tabulated in Table III.

SUMMARY AND CONCLUSIONS

1. The roentgen findings produced in the chest by five collagen diseases have been presented.

2. Selected case reports are given to illustrate how an analysis of various combinations of nonspecific roentgen signs may lead one to suspect a collagen disease, and occasionally make a specific diagnosis.

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ROENTGEN DIAGNOSIS OF ANOMALOUS PULMONARY VENOUS DRAINAGE OF RIGHT LUNG INTO INFERIOR VENA CAVA*

REPORT OF THREE NEW CASES

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ROENTGENOGRAPHY plays a significant role in the diagnosis of anomalous pulmonary venous drainage of the right lung into the inferior vena cava. Indeed, the anomaly can usually be recognized after inspection of the conventional frontal roentgenogram which reveals a characteristic multiple-branching vessel increasing in breadth from above downward to merge into a broad, crescentic channel adjacent to the right cardiac border. In contrast, the left lung appears well vascularized, even plethoric, and often larger than its mate. The right atrium, because of increased blood flow, is unduly prominent and often, also because of displacement of the heart by the enlarged left lung, extends well into the right hemithorax. Angiocardiography establishes the definitive diagnosis by clearly visualizing the anomalous right pulmonary vein plunging below the diaphragm at the cardiohepatic angle. Reopacification of the right atrium, ventricle and pulmonary arterial system confirms the existence of a left-right cardiac shunt.

Anomalous pulmonary venous drainage of the right lung into the inferior vena cava is rare. Prior to the advent of angiocardiography, the diagnosis was made only at necropsy or operation. Brody[2] in 1942, in a classic review of the pathologic literature found only 2 reports of anomalous pulmonary venous drainage into the inferior vena cava. The first living patient with this anomaly, diagnosed by angiocardiography in 1943, and another case studied with cardiac catheterization, were reported in 1949.[4] A recent paper reviewed the findings in 18 cases collected from the literature and added 3 new ones.[8] Other cases,[7,11] a series reported by Bruwer[3] and 3 cases recently reported from this center[18] bring the total number to well over 30.

Corrective surgery has been successful in 2 cases with anomalous pulmonary venous connection into the inferior vena cava complicated by atrial septal defects.[9] Three cases recently described with aberrant arterial branches from the abdominal aorta inserting into the right lower lobe were associated with dyspnea and chronic bronchopulmonary disease requiring right pneumonectomy.[8]

The purpose of this paper is to report 3 new cases of anomalous pulmonary venous drainage of the right lung into the inferior vena cava, each with an unusual feature; to review the experience of 9 cases studied at this center; and to re-emphasize the importance of roentgenography in diagnosis.

REPORT OF CASES

CASE 1. *Insertion of Right Pulmonary Veins into Inferior Vena Cava, Left Diaphragmatic Hernia.* A fifty-five year old housewife (N.Y.H. No. 739478) was referred by Dr. Laurence Miscall on May 24, 1956. The patient stated that she had been well until six months prior to admission when dyspnea requiring three pillows during sleep began. Gastrointestinal roentgen studies at another hospital disclosed a left hiatus hernia. Following gallbladder studies, a large mass in the right lower lung field was noted. Chest roentgen studies disclosed a left hiatus hernia and multiple branching vessels entering a large crescentic channel in the right

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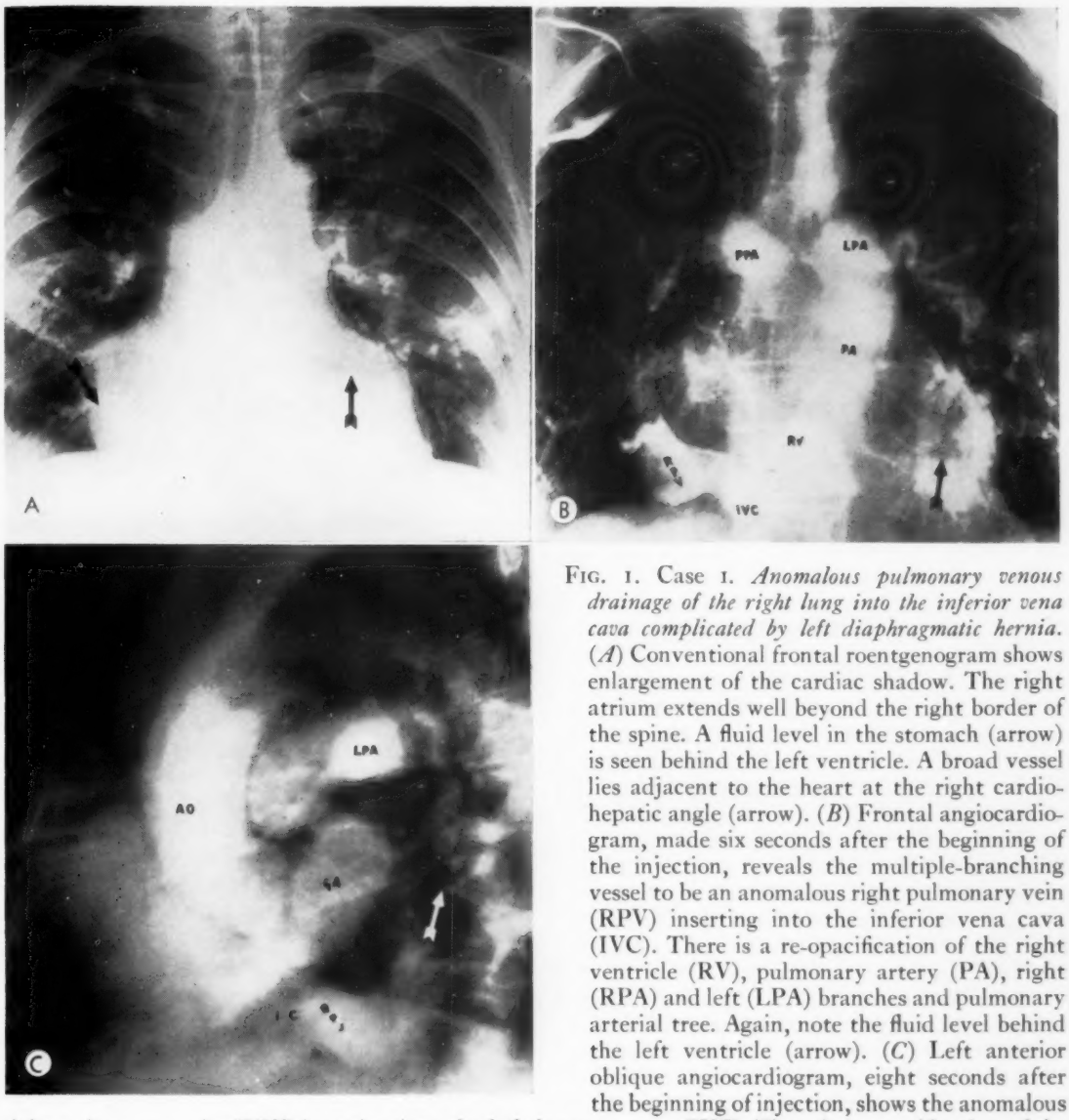

FIG. 1. Case 1. *Anomalous pulmonary venous drainage of the right lung into the inferior vena cava complicated by left diaphragmatic hernia.* (*A*) Conventional frontal roentgenogram shows enlargement of the cardiac shadow. The right atrium extends well beyond the right border of the spine. A fluid level in the stomach (arrow) is seen behind the left ventricle. A broad vessel lies adjacent to the heart at the right cardiohepatic angle (arrow). (*B*) Frontal angiocardiogram, made six seconds after the beginning of the injection, reveals the multiple-branching vessel to be an anomalous right pulmonary vein (RPV) inserting into the inferior vena cava (IVC). There is a re-opacification of the right ventricle (RV), pulmonary artery (PA), right (RPA) and left (LPA) branches and pulmonary arterial tree. Again, note the fluid level behind the left ventricle (arrow). (*C*) Left anterior oblique angiocardiogram, eight seconds after the beginning of injection, shows the anomalous right pulmonary vein (RPV) inserting into the inferior vena cava (IVC). There is re-opacification of the right cardiac chambers and pulmonary artery, especially the left branch (LPA). The left atrium (LA) and aorta (AO) are also well opacified (the left ventricle is in contraction). Arrow points to the air outlining the upper wall of the gastric hernia.

cardiophrenic region (Fig. 1*A*). Angiocardiography,* in frontal and left anterior oblique views, revealed a huge pulmonary vein draining the right lung and converging towards the right lower cardiac area in the region of the inferior vena cava (Fig. 1, *B* and *C*). The right atrium was enlarged and extended beyond the right border of the spine. The right ventricle, pulmonary artery and left branch were also enlarged. Recirculation of contrast material into the right cardiac chambers and pulmonary artery was also seen.

Physical examination showed the patient to be obese, acyanotic and in no discomfort. The heart was enlarged, the blood pressure was 130/84 mm. Hg. A systolic murmur was heard over the precordium, most prominent at the base of the heart, extending along the right side

* Performed by Dr. J. E. Goldstein, Director and Attending Roentgenologist, Jamaica Hospital, Queens, New York.

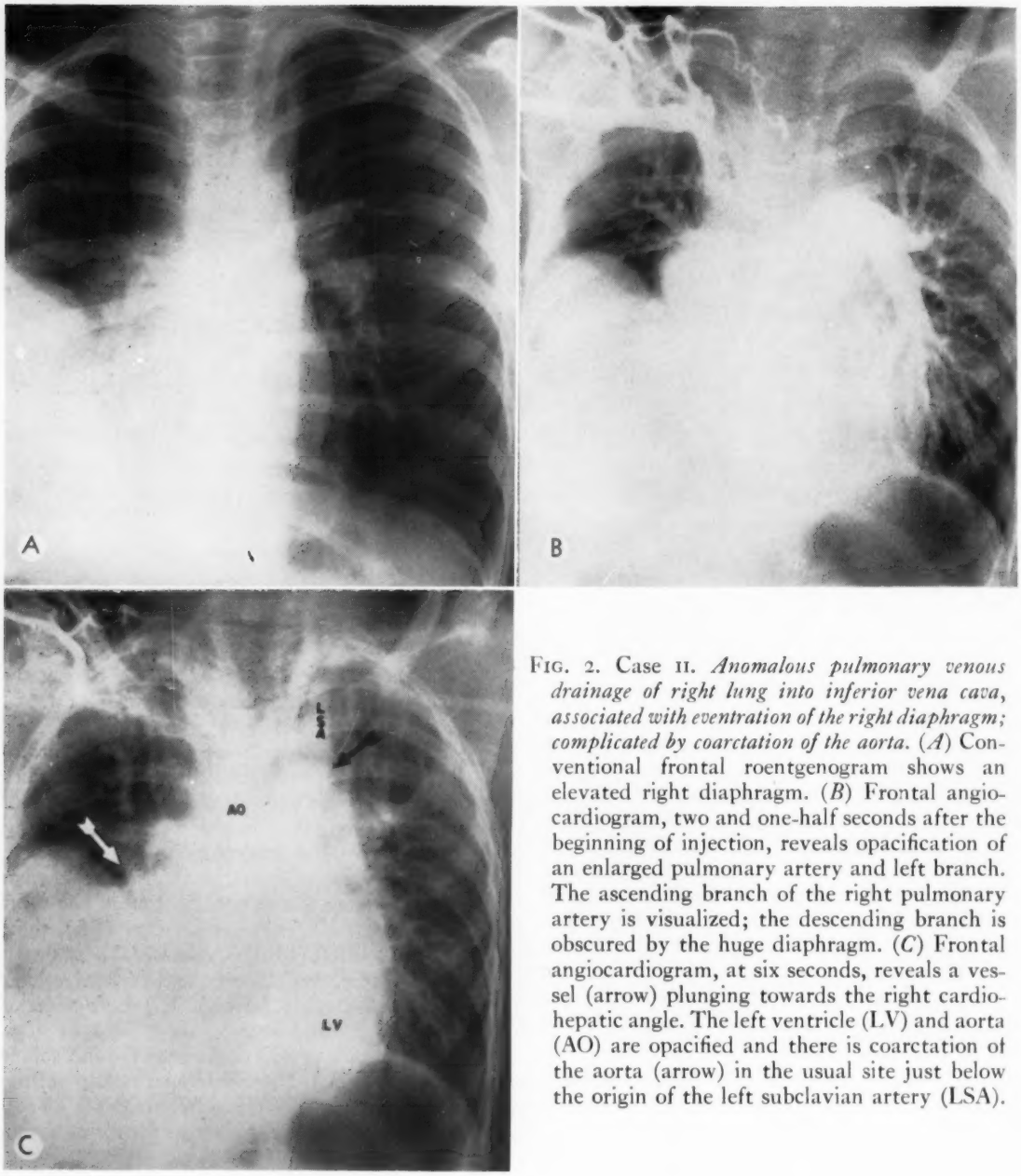

FIG. 2. Case II. *Anomalous pulmonary venous drainage of right lung into inferior vena cava, associated with eventration of the right diaphragm; complicated by coarctation of the aorta.* (*A*) Conventional frontal roentgenogram shows an elevated right diaphragm. (*B*) Frontal angiocardiogram, two and one-half seconds after the beginning of injection, reveals opacification of an enlarged pulmonary artery and left branch. The ascending branch of the right pulmonary artery is visualized; the descending branch is obscured by the huge diaphragm. (*C*) Frontal angiocardiogram, at six seconds, reveals a vessel (arrow) plunging towards the right cardiohepatic angle. The left ventricle (LV) and aorta (AO) are opacified and there is coarctation of the aorta (arrow) in the usual site just below the origin of the left subclavian artery (LSA).

of the sternum. The lungs were clear. Laboratory data revealed: erythrocytes, 4,500,000 per cu. mm.; hemoglobin, 12.8 gm.; leukocytes, 7,500. The electrocardiogram showed normal sinus rhythm, deviation of the electrical axis to the left and a horizontal position of the heart.

Cardiac catheterization* showed an increased oxygen content of the right atrial blood (15.4 vol. per cent), whereas the superior vena caval blood oxygen content was 13.5 vol. per cent and the inferior vena caval 14.7 vol. per cent, compared to the brachial artery oxygen content of 17.4 vol. per cent. These findings indicated a left to right shunt of 2.59 l./min. at the atrial level comprising only 30 per cent of the pulmonary blood flow which was 4.93 l./min./m.^{2}. This was also less than 50 per cent of the normal systemic blood flow (5.7 l./min. and 3.39 l./min./m.^{2}). Methylene blue injections into the right atrium and ventricle revealed the cir-

* Performed by Dr. Daniel S. Lukas.

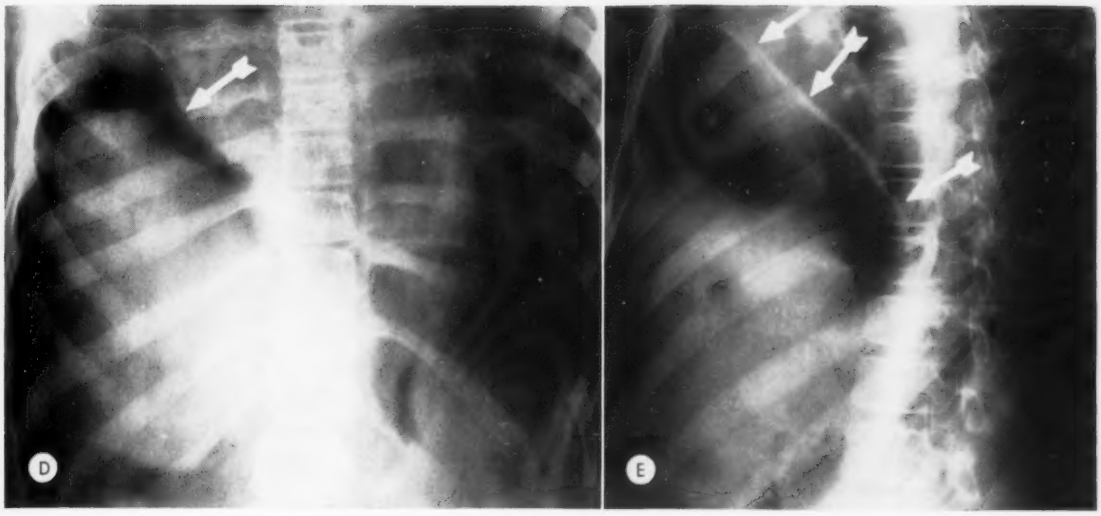

FIG. 2. Case II. (*D*) Erect, frontal pneumoperitoneal roentgenogram showing the elevated thin right diaphragm (arrow) well above the liver and apparently adherent to the inferior border of the heart. (*E*) Left oblique, erect, pneumoperitoneal roentgenogram showing eventration of the right diaphragm (arrows).

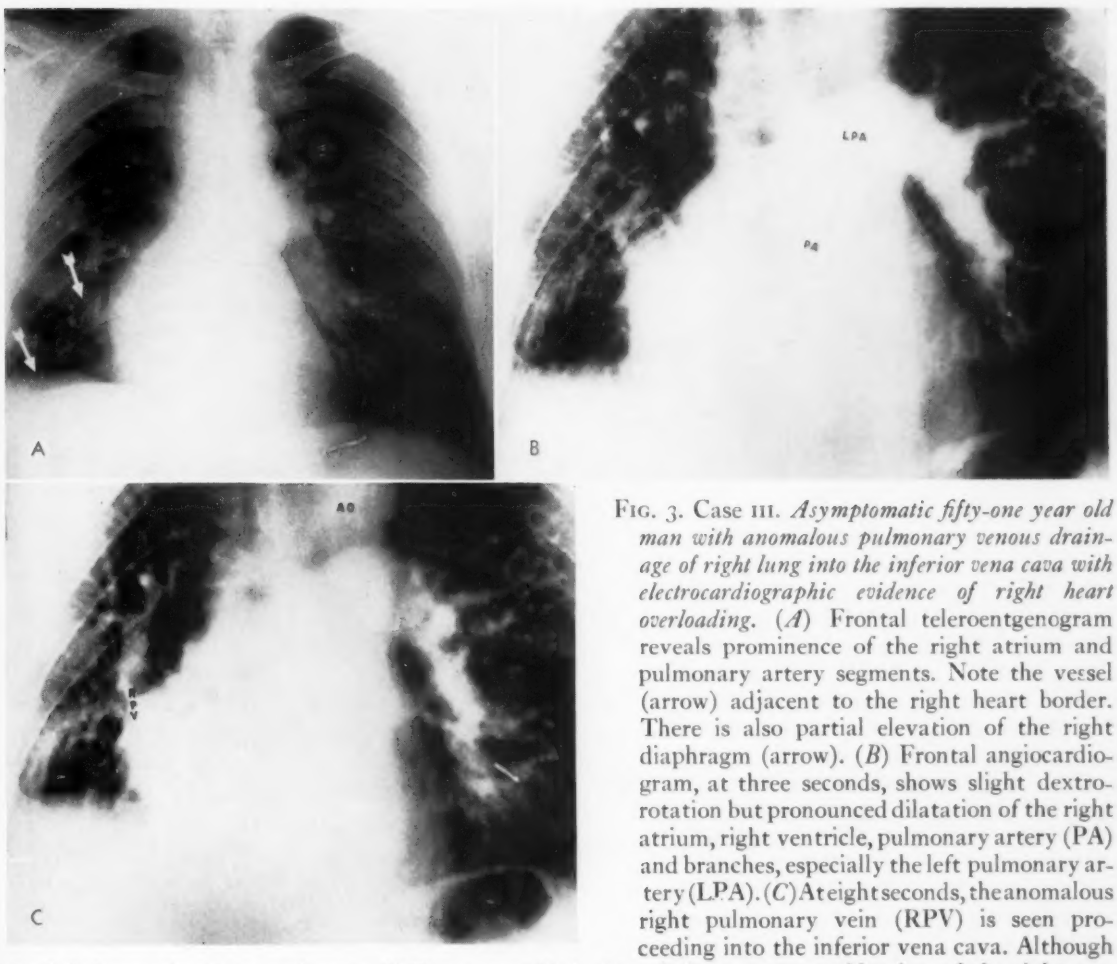

FIG. 3. Case III. *Asymptomatic fifty-one year old man with anomalous pulmonary venous drainage of right lung into the inferior vena cava with electrocardiographic evidence of right heart overloading.* (*A*) Frontal teleroentgenogram reveals prominence of the right atrium and pulmonary artery segments. Note the vessel (arrow) adjacent to the right heart border. There is also partial elevation of the right diaphragm (arrow). (*B*) Frontal angiocardiogram, at three seconds, shows slight dextrorotation but pronounced dilatation of the right atrium, right ventricle, pulmonary artery (PA) and branches, especially the left pulmonary artery (LPA). (*C*) At eight seconds, the anomalous right pulmonary vein (RPV) is seen proceeding into the inferior vena cava. Although the left heart chambers and aorta (AO) are opacified, there is intense re-opacification of the right cardiac chambers, pulmonary artery and pulmonary arterial tree.

culation times from these structures to be normal, thereby excluding the presence of an atrial shunt. The pulmonary artery pressure could not be secured. The right ventricle pressure was slightly elevated, 34/8 mm. Hg (normal, 25/2). The right atrial pressure was also slightly elevated, 8 mm. Hg (normal, 5). Following these studies, the patient was returned to Jamaica Hospital, and the hiatal hernia was repaired by Dr. Laurence Miscall. The patient made an uneventful recovery. Recent follow-up examination found her well and asymptomatic.

CASE II. *Right Pulmonary Veins Draining into Inferior Vena Cava, Dextrorotation of the Heart, Eventration of the Right Diaphragm, and Coarctation of the Aorta.* A ten year old schoolgirl (N.Y.H. No. 488547) was first seen in the clinic at the age of three years because of recurrent respiratory infections. A cardiac murmur and a deformed thorax resulted in requests for chest roentgenographic and electrocardiographic examinations. When these were seen, she was referred to the hospital for study. Physical examination revealed undue prominence of the upper sternum. General nourishment, development and weight were normal. Soft systolic murmurs were heard at the apex and pulmonic areas of the heart. The arm blood pressure was 124/80 mm. Hg; leg pressures were not recorded and mention of lower extremity pulsation was not made. Elevation of the right diaphragm was noted in the roentgenograms and the electrocardiogram revealed deviation of the electrical axis to the left. For the next six years the patient visited the clinic periodically owing to repeated respiratory infections. In 1950, blood pressure readings were: right arm, 116/82; right leg, 152/120 mm. Hg. However, in 1953, leg pressures and pulsations could not be obtained and coarctation of the aorta was suspected.

On April 4, 1954, the patient was again hospitalized and physical examination showed her to be well developed and nourished. Blood pressure reading in the right arm was 125/60 mm. Hg, pressures in the legs could not be obtained. The sternum was prominent and fullness and bulging of the right thorax were seen. A loud systolic (Grade 3) murmur was heard over the left precordium, loudest along the upper left sternum. The second pulmonic sound was accentuated and split. The lungs were clear but dullness to flatness was present in the lower three fourths of the right thorax.

The chest roentgenogram (Fig. 2*A*) revealed a high right diaphragm and a small right lung. Angiocardiograms were made on April 23, 1956, and disclosed a hypoplastic right lung, normal pulmonary arterial circulation of the right upper lobe, and crowding with scant pulmonary arterial circulation of the remaining portion of the right lung (Fig. 2*B*). There was dextrorotation of the heart and at the time of left atrial, ventricular and aortic filling, a large vessel, an anomalous pulmonary venous channel, was seen plunging below the eventrated diaphragm (Fig. 2*C*). Re-opacification of the right atrium, ventricle and pulmonary artery and branches was also present. In addition, coarctation of the aorta, at the usual site, was clearly visualized (Fig. 2*C*). Subsequently, pneumoperitoneal studies disclosed a high, fixed, atrophic, right diaphragm (Fig. 2, *D* and *E*).

At another hospital in September, 1957, the right chest was explored, and an extremely high attachment of the right diaphragm along the lateral border was found. The pericardium along the right inferior heart border and the inferior vena cava were short and adherent to the drawn-up diaphragm. Because of the firm and high attachments of these structures it was impossible to dislocate the diaphragm downward. Palpation of the right pulmonary artery disclosed markedly diminished pulsations, confirming the presence of a hypoplastic pulmonary arterial circulation. The lung appeared to have the normal lobar arrangements, but the course of the right pulmonary vein into the inferior vena cava could not be followed on account of the dense diaphragmatic, pericardial and inferior vena caval adhesions. Because of the absence of hypertension, resection of the coarctation was postponed.

CASE III. *Anomalous Pulmonary Venous Drainage of the Right Lung into the Inferior Vena Cava in a Fifty-one Year Old Asymptomatic Man with Electrocardiographic Changes Suggesting Right Ventricular Overloading or Atrial Septal Defect.* A conventional frontal roentgenogram, taken routinely on July 23, 1957, as part of a yearly physical examination in a fifty-one year old asymptomatic executive, disclosed the characteristic features of anomalous pulmonary venous drainage of the right lung into the inferior vena cava (Fig. 3*A*).

Physical examination was entirely normal. There were no cardiac murmurs, the pulse rate was 75 and the blood pressure was 125/80 mm. Hg. The electrocardiogram showed deviation of the electrical axis to the right with incomplete bundle branch block, the typical RSR^1V_1 pattern due to right ventricular overloading.[1,12]

Angiocardiography made serially, on September 18, 1957, disclosed enlargement and slight dextrorotation of the right atrium, ventricle and pulmonary artery (Fig. 3*B*). The right pulmonary artery was smaller than the left with crowding and distortion of the pulmonary arterial circulation of the right lower lung (Fig. 3*B*). When the left heart structures were visualized, a large multiple branching pulmonary vein was seen along the right cardiac border proceeding into the inferior vena cava (Fig. 3*C*). The left atrium, ventricle and aorta appeared to be normal in size and contour. During the time of left heart filling, there was re-opacification of the right heart structures (right atrium, ventricle, pulmonary artery and pulmonary arterial tree). Cardiac catheterization, bronchography and bronchoscopy were recommended but declined by the patient.

DISCUSSION

Physiologic and Anatomic Features. The embryologic features of anomalous pulmonary venous drainage have recently been reviewed[6] and will not be further discussed except to point out that the development of the superior vena caval system, persistence of the left cardinal vein (left superior vena cava) and malformations of the pulmonary veins are closely related. Two types of anomalous pulmonary venous drainage are recognized. The partial includes insertions of pulmonary veins into the right atrium or its venous tributaries from a lobe or lung. It is occasionally an incidental finding at operation or autopsy and, unless complicated by an associated cardiovascular defect, is compatible with long life. In contrast, complete transposition of the pulmonary veins may produce serious symptoms very early in life; long survival has been reported only occasionally. In the total type, an atrial septal defect must also exist to sustain life.[14]

The diagnosis of anomalous insertion of pulmonary veins into the inferior vena cava may be established when cardiac catheterization studies reveal a higher oxygen saturation of the inferior vena caval blood than in that of the superior vena cava and right atrium. An associated atrial septal defect can be demonstrated by advancing the catheter from the right into the left atrium and by circulation time determinations with methylene blue.[14] In 8 cases studied at this center, cardiac catheterization was necessary in only 2 instances. It was done in order to establish the identity of the lesion in one case;[4] in the other, because an atrial defect was suspected in a dyspneic patient with diaphragmatic hernia (Case 1). Cardiac catheterization was also advised in Case III in order to determine the presence of an atrial septal defect. The electrocardiographic evidence of right heart overloading (right axis deviation and the RSR^1V_1 pattern) and the unusually large main pulmonary artery also suggested pulmonary hypertension. However, a left to right shunt, if large enough, can cause similar electrocardiographic abnormalities. Cardiac catheterization is usually unnecessary in the partial types of anomalous pulmonary venous drainage and should be reserved for symptomatic patients suspected of having complicating lesions such as atrial septal defects.[14]

Anomalies of the right pulmonary arterial system, lung or both have long been recognized in patients with anomalous pulmonary venous drainage into the inferior vena cava. The sparsity of the pulmonary circulation in the right upper lung field and the unusual configuration of the vessels in the lower two thirds of the right lung field were noted in the first publication.[4] Halasz, Halloran and Liebow[8] have also demonstrated hypoplasia of the right pulmonary artery in 3 cases by making beautiful latex casts of the pulmonary circulation of the excised specimen.

Recently, Bruwer[3] reported bronchoscopic and bronchographic abnormalities

of the right lung of a patient with anomalous pulmonary venous connection into the inferior vena cava and suggested that hypogenesis or agenesis of a portion of the right lung may occur regularly. Halasz *et al.*[8] with special injection techniques also showed abnormal arrangement of the bronchi and occurrence of bronchial diverticula of the right lung in their cases. Since the majority of our cases were asymptomatic, bronchography and bronchoscopy were not advised.

Pleural, pericardial and diaphragmatic abnormalities apparently also occur in the right chest of patients with pulmonary venous connection into the inferior vena cava. Pleuropericardial adhesions were encountered in Case II and in another patient after exploratory thoracotomy.[18] The entire right diaphragm appeared elevated in Case II (Fig. 2), whereas in Case III (Fig. 3) only slight and partial elevation of the right diaphragm was seen. In a case recently reported[18] partial elevation of the posterior leaf of the right diaphragm simulated a thoracic tumor. Drake and Lynch[5] also encountered a diaphragmatic anomaly on exploration of the chest of a patient with anomalous pulmonary venous drainage of the right lung into the inferior vena cava.

Cardiac abnormalities, acquired and congenital, also occur with anomalous pulmonary venous drainage of the right lung into the inferior vena cava. As a result of the left to right shunt, the right atrium is enlarged and often projects well beyond the right border of the spine. The plethoric overdistended left lung exerting pressure on the hypoplastic right lung is also responsible for the cardiac displacement into the right hemithorax. The marked dextrorotation of the heart found in the 3 cases recently described[18] was apparently not due to dilatation and displacement of cardiac chambers alone. Rather, it appeared to be an associated, probably congenital, malformation.[17] Postmortem study to determine whether the dextrorotation of the heart is an independent embryologic anomaly related to uncomplicated dextroversion is yet to be secured. Also, extensive pleuropericardial adhesions may play a role in producing cardiac rotation in some cases.

Clinical Features. Six of 9 patients with anomalous pulmonary venous drainage of the right lung into the inferior vena cava studied at this center were asymptomatic and discovered after routine chest roentgenography. For example, one patient, a naval officer, was found to have hypertension on a prepromotion examination, and after the routine chest roentgenographic examination was suspected of having the anomaly.[4] Another patient (Case I), had dyspnea and a huge left diaphragmatic hernia. Study of the chest roentgenogram disclosed the right-sided pulmonary venous anomaly, which was confirmed by angiocardiography. Cardiac catheterization excluded the presence of an atrial septal defect. The diaphragmatic hernia was thought to cause the dyspnea and so it proved, for repair of the diaphragm completely relieved the symptom. Angiocardiography, performed because of the suspicion of aortic coarctation (Case III), led to the discovery of an anomalous right pulmonary vein. The ages in 9 cases varied from ten to fifty-nine years. The youngest was ten years, 3 were under forty years and 5 were over fifty years. Five were male and 4 female. Only one was a Negro.

Heart murmurs were present in 3 patients and were usually systolic, of low intensity (Grade 2), located at the base of the heart. In 3 patients with dextrorotation of the heart, the heart sounds were more pronounced to the right of the sternum. Electrocardiographic studies in all but one patient were normal. In that case, right axis deviation and the RSR^1V_1 pattern suggested right ventricular overloading (Case III).[1,12] Two patients with moderate hypertension had slight deviation of the electrical axis to the left. The 3 patients with rotation of the heart into the right

hemithorax showed no abnormalities of the right precordial tracings.

Roentgenography. A common feature, observed in all but one case of anomalous pulmonary venous drainage of the right lung into the inferior vena cava, is the appearance in the conventional frontal roentgenogram of a broad, multiple-branching channel curving in crescentic fashion near the right cardiac border and descending toward the right cardiohepatic region. Often, the right lung is hypoplastic with narrowed intercostal spaces while the left lung is voluminous and plethoric. In 5 instances the right atrium was prominent and projected well into the right hemithorax. The cases with pronounced rotation of the heart into the right hemithorax also had the characteristic vascular deformity adjacent to the right cardiac silhouette.[18] In one patient, the rotated heart obscured the anomalous pulmonary venous vessels; however, an overpenetrated film showed the vascular anomaly. In Case 11, the high diaphragm precluded detection of the characteristic right lower pulmonary venous channel. Laminagraphy has been highly recommended for diagnosis of anomalous pulmonary venous drainage of the right lung into the inferior vena cava.[3] Although the findings are informative, laminagraphy cannot make the definitive diagnosis.

Angiocardiography, by visualizing the course of the right anomalous pulmonary venous system as it enters the inferior vena cava, provides the definitive diagnosis of right pulmonary venous drainage into the inferior vena cava (Fig. 1, *B* and *C;* 2*C* and 3*C*). In addition, it gives information regarding the cardiac chambers, pulmonary arteries and pulmonary arterial vasculature (Fig. 2*B* and 3*B*). The size of the chambers, the degree of cardiac rotation and abnormality of the pulmonary arterial system are revealed. Recirculation of blood from the anomalous pulmonary venous system into the right heart structures can also be demonstrated (Fig. 1, *B* and *C;* 2*C* and 3*C*). Arteries from the abdominal aorta inserting into the right lower lobe were demonstrated angiocardiographically by Halasz *et al.*[8] in one case.

Differential Diagnosis. A pulmonary arteriovenous fistula in the right lower lung field may simulate anomalous right pulmonary veins inserting into the inferior vena cava.[15] However, normal right lung pulmonary vasculature, except at the site of the aneurysm, absence of right atrial prominence and the presence of a vascular bruit should easily differentiate the lesion. An anomalous pulmonary venous trunk may sometimes be obscured by a rotated heart. In such a situation, congenital absence of the right pulmonary artery may come to mind because of the mediastinal and cardiac shifting.[13] Absence of the right pulmonary arterial tree, however, and the "lacy" pattern of the vasculature should suggest the bronchial rather than the pulmonary arterial circulation. Diminished breath sounds due to hypoplasia of the right lung and physical and roentgen signs of overdistention of the opposite lung will also give clues to the diagnosis. Finally, a patient with anomalous systemic arteries from the abdominal aorta perforating the diaphragm and entering a hypoplastic right lung may, because of cardiac dextrorotation and right pulmonary hypoplasia, be difficult to distinguish from anomalous right pulmonary veins draining into the inferior vena cava.[16] Clinically, in such a case, disabling dyspnea may be present.[10] Without angiocardiography it would indeed be difficult to establish the diagnosis.

SUMMARY AND CONCLUSIONS

Three new patients with anomalous pulmonary venous drainage of the right lung into the inferior vena cava had unusual complications as compared to 6 reported asymptomatic cases studied at this center. In the first, a huge left diaphragmatic hernia caused dyspnea in a fifty-five year old woman. Cardiac catheterization re-

vealed an intact atrial septum. Surgical repair of the hernia resulted in the patient becoming asymptomatic. A ten year old patient, the youngest in our series, had an unusually high, immobile right diaphragm and coarctation of the aorta. Angiocardiography, performed in order to visualize the aortic coarctation, revealed an unsuspected pulmonary venous channel draining into the inferior vena cava. Exploratory thoracotomy confirmed many of the roentgen findings. Finally, an asymptomatic fifty year old man was found to have the characteristic roentgen features of the anomaly. However, electrocardiography revealed overloading of the right ventricle, an enlarged pulmonary artery and a plethoric left lung that suggested an atrial septal defect complication. When dyspnea and bronchopulmonary disease are present in patients with anomalous drainage of the right lung into the inferior vena cava, abdominal aortic branches inserting into the right lower lobe should be suspected. Right pneumonectomy in such cases has been curative.

Asymptomatic patients with partial anomalous drainage of the right lung into the inferior vena cava do not require treatment, this type of anomaly being compatible with long life. However, patients with dyspnea, recurrent pulmonary infections and plethora of the lungs may have an associated cardiac lesion, such as an atrial septal defect that requires repair. Cardiac catheterization in order to assess the amount of the shunt and the degree of damage to the right ventricle (hypertension) is recommended in such circumstances. Present day open heart surgery may be expected to close the atrial septal defect and correct the pulmonary venous anomaly.

The conventional frontal roentgenogram of the chest often discloses the characteristic features of anomalous pulmonary venous drainage of the right lung into the inferior vena cava. Occasionally, a high right diaphragm or dextroversion of the heart may obscure the anomalous pulmonary venous channel at the right cardiohepatic angle. Angiocardiography, however, not only confirms and establishes the diagnosis, but may, in some instances, reveal additional anomalies such as abnormalities of the right pulmonary artery and abdominal aortic branches to the right lower lobe.

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ADDENDUM

Since this paper was submitted for publication, 2 additional cases of anomalous pulmonary venous drainage of the right lung into the inferior vena cava have been published: (1) WOOD, J. C., CONRAD, M., and MORROW, A. G. Partial anomalous venous connection: a case report illustrating diagnostic techniques. *Am. Heart J.*, 1957, *54*, 422–428; and (2) BENDER, VON F., HILGENBERG, F., and JUGEHÜLSING, G. Dextrokardie mit Pulmonalvenentransposition bei partieller Lungenagenesie. *Ztschr. Kreislaufforsch.*, 1957, *46*, 127–179. The latter report also emphasizes dextrorotation of the heart that may occur in this type of partial pulmonary venous anomaly.

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UNUSUAL ROENTGEN MANIFESTATIONS OF PRIMARY STAPHYLOCOCCAL PNEUMONIA IN INFANTS AND YOUNG CHILDREN*

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PRIMARY staphylococcal pneumonia is defined as staphylococcal pneumonia arising in the absence of pre-existing pulmonary disease; it has become a common and important entity. A number of authors have expressed the belief that the prevalence of the disease is increasing.[1,2,8] Further evidence of this is shown in Table 1, where the number of authenticated cases seen at The Johns Hopkins Hospital in 1955, 1956 and 1957 are compared with those observed in 1944 and 1945, and in 1951 and 1952.

This paper reports 41 cases of staphylococcal pneumonia seen at this hospital during the period between 1955 and 1957 inclusive. Of these, 7 demonstrated one or more unusual roentgen manifestations which appear of particular interest. All cases were proved by identification of the hemolytic *Staphylococcus aureus* in cultures obtained from pleural fluid, blood or the lung itself. The presence of pathogenic staphylococci in nose and throat secretions was not considered adequate to establish the diagnosis.

The increase in prevalence of the disease has fortunately been accompanied by a substantial decrease in mortality. This probably has been mainly due to prompt specific antibiotic therapy. Many of the commonly used antibiotics, however, are frequently ineffective. Hence, prompt etiologic diagnosis is of great importance if effective therapy is to be started early. Clinically, this often cannot be achieved because of misleading physical signs and uncertain bacteriologic proof.

Fortunately, staphylococcal pneumonia, especially in children less then one year of age, is one of the few lung diseases in which early roentgen manifestations appear to have pathognomonic significance. The reliability of these signs has been emphasized by several investigators.[1,4,5,6]

TYPICAL ROENTGEN FEATURES

1. *Localized Parenchymal Infiltrations.* These are often of lobar distribution and involve one to three lobes of the same side[1] (Fig. 1). Bilateral involvement occurred only 5 times in our series of 41 cases and in 4 of these it was minimal and transient. Several recent cases suggest that, as in adults, it may be more common in older children. Mediastinal lymph node enlargement is unusual.

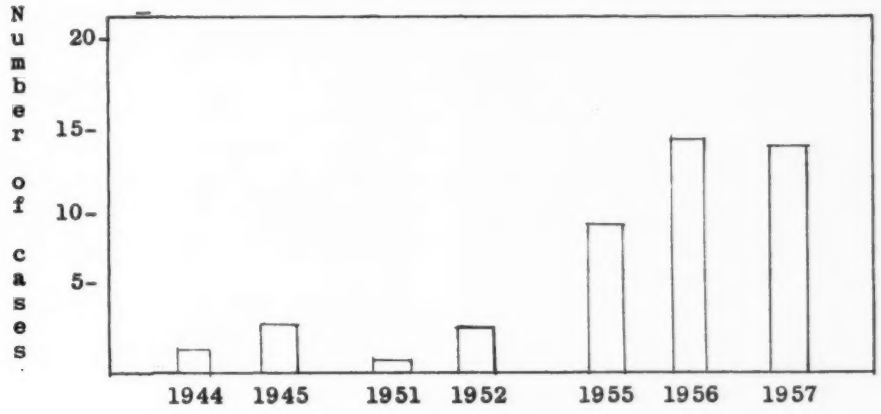
2. *Pleural Involvement.* Most frequently this takes the form of an empyema. In our series empyema was present in approximately 90 per cent of the cases. We also observed spontaneous pneumothorax and pyopneumothorax, often of the tension types, in approximately 25 per cent of our patients.

3. *Development of Pneumatoceles.* Pneumatoceles are emphysematous cysts or cavities probably arising from small areas of necrosis located in the peribronchial interstitial tissue. These areas communicate with bronchi through small sinuses and are inflated rapidly by check valve action of the edematous bronchial mucosa or luminal secretions.[1,4,7] They usually form in the most densely infiltrated areas, are thin-walled and sometimes contain fluid. During the acute stage of the disease they often, within hours, change considerably in number, size and position (Fig. 1). While they may be as small as 0.5 cm. in diameter, they may expand or coalesce to form very large cysts indistinguishable

* Presented at the Fifty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 1–4, 1957.

Table I

CASES/YEAR—PRIMARY STAPHYLOCOCCAL PNEUMONIAS



from and producing effects similar to tension pneumothorax. This is especially true if they are situated anteriorly.

Marked parenchymal and pleural change may remain after complete clinical recovery from the disease; usually, however, this disappears within two weeks to two months and corrective surgery should not be undertaken prematurely. The average period from onset to complete healing of the disease in our experience has been approximately four weeks. Not infrequently, residual, asymptomatic pneumatoceles persist for longer periods; we have followed a few for periods of six to eight months. Since they usually exhibit slow, spontaneous diminution in size and final disappearance, they should not be removed surgically.[1] Many so-called "congenital cysts" in infants are probably acquired in this way.[3]

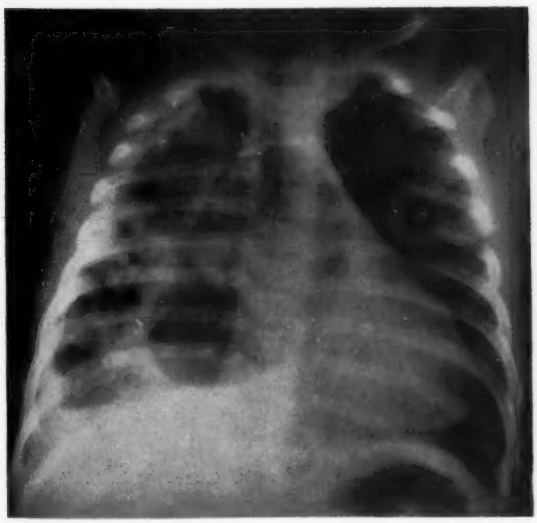


Fig. 1. Right sided staphylococcal pneumonia. Parenchymal infiltrations of all the lobes. A large anterior pneumatocele is displacing the heart and mediastinum to the left. Numerous smaller pneumatoceles are present lateral to it. Pleural fluid is beginning to form along the right lateral chest wall.

DIFFERENTIAL DIAGNOSIS

Bilateral and often generalized pneumonic infiltrations are common in infants less then one year of age and usually do not suggest a specific etiology. On the other hand, when localized unilateral infiltrations are present, especially when these are associated with pleural changes, staphylococcal pneumonia should be primarily suspected. A similar picture is in our experience quite frequently associated with primary tuberculous pneumonia and is occasionally present in pneumococcal and nonspecific pneumonias. It has also been described in *Hemophilus influenzae* Type B pneumonia. However, the coincident formation of pneumatoceles appears very characteristic of staphylococcal pneumonia. Since the beginning of 1955, we have seen only one case each of tuberculous, pneumococcal and pyocyanial pneumonia which exhibited shadows indistinguishable from those produced by pneumatoceles. In each case the

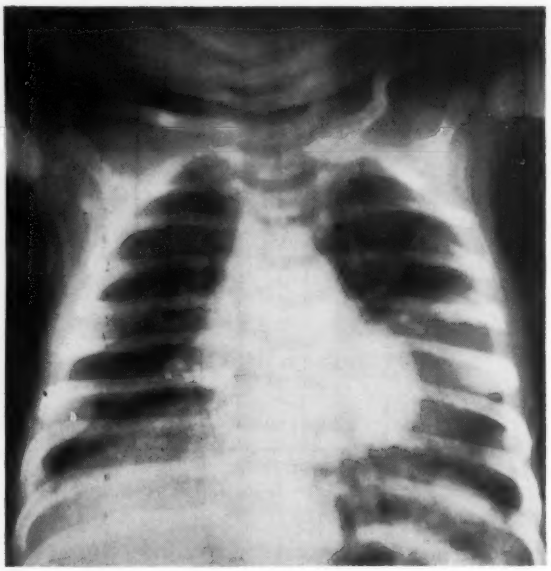


Fig. 2. Case I. Spheroid consolidation in the superior portion of the left upper lobe. Note the absence of pleural involvement.

"pneumatocele" was single and persisted unchanged for several days.

When spontaneous pneumothorax or pyopneumothorax *per se* is present, staphylococcal pneumonia should likewise be primarily suspected.[4,6]

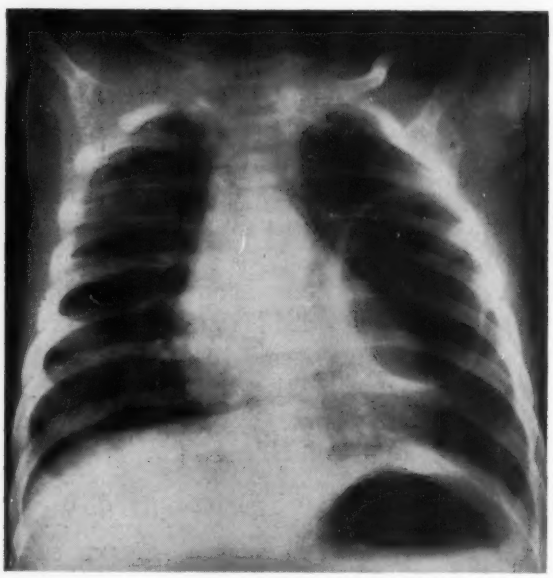


Fig. 3. Case I. A large thin-walled pneumatocele has formed at the site of the preceding spheroid consolidation by coalescence of multiple small pneumatoceles. It required six to seven months to resolve.

UNUSUAL MANIFESTATIONS

In a number of cases (approximately 15 per cent) the early picture was atypical and the roentgen diagnosis of staphylococcal pneumonia was not made at the onset. The unusual features were: unilateral and bilateral spheroid lesions; early nonspecific changes; hilar lymph adenopathy and massive atelectasis. These cases are described in some detail.

REPORT OF CASES

Case I. Figures 2 and 3 show the unusual course of a staphylococcal pneumonia in a one month old female. Approximately three weeks before the first chest roentgenogram was taken, she was treated at The Johns Hopkins Hospital for peri-orbital cellulitis, which responded to penicillin. The first roentgenogram illustrated diffuse infiltration of most of the left lung, the suggestion of a small pneumatocele and no pleural involvement. In the course of the following two weeks the diffuse infiltration diminished to a spheroidal lesion, approximately 4 cm. in diameter, situated in the superior portion of the left upper lobe (Fig. 2). Subsequently, multiple small pneumatoceles appeared within this area and coalesced to form one large, thin-walled pneumatocele (Fig. 3). At this time the patient was asymptomatic. In the next six months the pneumatocele decreased spontaneously to a diameter of 1.5 cm. and later disappeared.

Case II. Four weeks following pylorotomy, the patient, a one month old male, was admitted with clinical signs of bilateral pneumonia. Both bases exhibited symmetric spheroid lesions about 3 cm. in diameter; pleural involvement was not present (Fig. 4). Multiple small pneumatoceles developed in these areas within two days. Subsequently the patient developed bilateral pneumothorax of which he died three days after the original lesions were discovered.

Case III. This two month old female was suspected clinically and roentgenologically to have severe bronchopneumonia and bronchiolitis (Fig. 5). The patient's condition rapidly worsened and a second roentgenogram taken approximately sixteen hours later (Fig. 6) showed some of the typical features of staphylococcal pneumonia. Specific antibiotics were immediately given in large doses. In spite of this, the patient died thirty-six hours after admission.

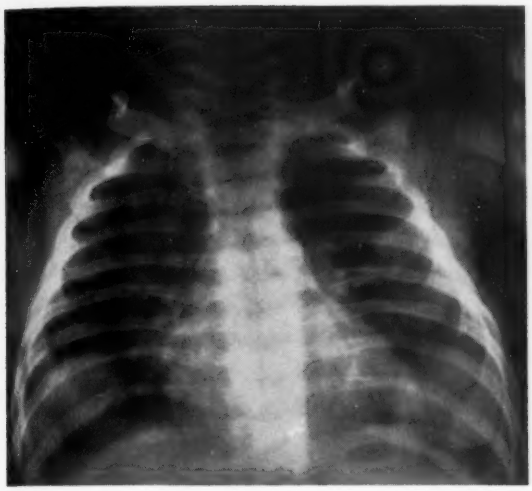


Fig. 4. Case II. Symmetric spheroid areas of consolidations in both bases. Again no pleural involvement is evident. Pneumatocele formation within these areas was followed by bilateral pneumothorax.

Case IV. This one and one-half month old male presented with clinical and roentgenologic signs of severe bronchiolitis and bronchopneumonia (Fig. 7). After a stationary course of seven days, the patient's condition rapidly began to deteriorate. Another chest roentgenogram was taken and the findings strongly suggested staphylococcal pneumonia (Fig. 8). The infant's conditon improved dramatically after the institution of specific antibiotic therapy and intercostal drainage. Recovery was uneventful.

Fig. 5. Case III. Bilateral hyperaeration associated with infiltrations in the right lower and probably also right upper lobes. The picture is very suggestive of the common and often nonspecific, diffuse bronchiolitis, associated with focal bronchopneumonia.

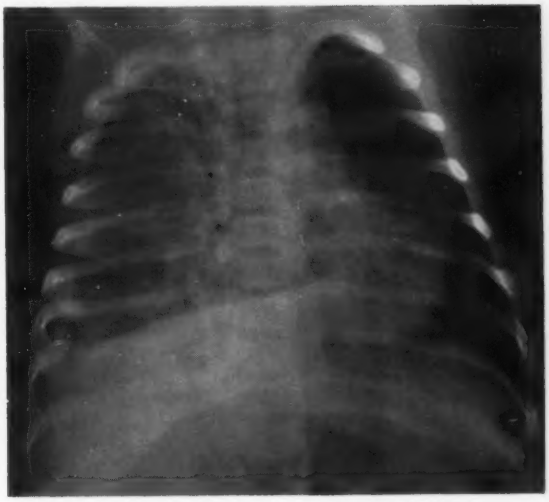


Fig. 6. Case III. Parenchymal infiltrates are present throughout the entire right lung, which is hyperexpanded displacing the heart and mediastinum to the left. Empyema fluid is beginning to form along the right lateral chest wall. This roentgenogram was taken shortly before the infant died, and only sixteen hours after the roentgenogram shown in Figure 5.

Case V. A two and one-half year old male was admitted with clinical evidence of extensive right-sided pneumonia. The admission

Fig. 7. Case IV. Bilateral hyperaeration and left basilar infiltrate very similar in appearance to that of Figure 5.

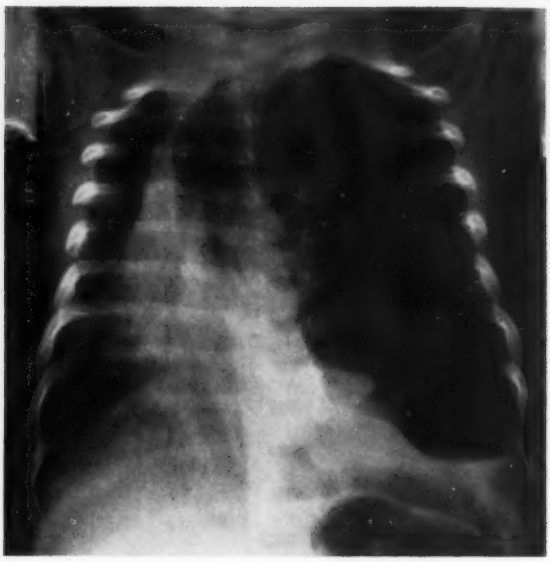


Fig. 8. Case iv. Roentgenogram taken one week after Figure 7 shows a massive tension pyopneumothorax or giant pneumatoceles displacing the heart and mediastinum markedly to the right.

chest roentgenogram showed marked hilar lymph node enlargement, perihilar infiltration and pleural effusion on the right (Fig. 9). Primary tuberculosis and fungus disease were entertained as etiologic agents, but tuberculin and fungus skin tests were negative. The lymph adenopathy cleared together with the parenchymal and pleural changes.

Case vi. A seventeen month old male suddenly developed severe dyspnea, cyanosis and moderate pyrexia. The chest roentgenogram

Fig. 9. Case v. Right hilar lymph adenopathy, perihilar infiltration and pleural fluid along the right lateral chest wall suggest primary tuberculous pneumonia or fungus disease with effusion. Staphylococci in pure culture were found in aspirated pleural fluid.

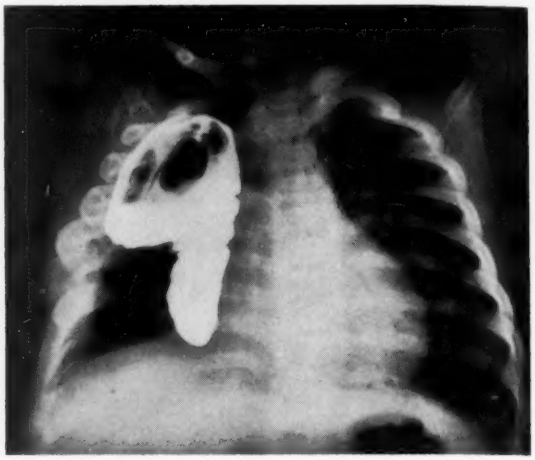


Fig. 10. Case vii. Dionosil instillation through a chest catheter revealed this extensive empyema cavity surrounding the right upper lobe and insinuating itself between the heart and the lower portion of the right lung. Roentgenologically only some pleural thickening along the right lateral chest wall was noted prior to the introduction of the contrast medium.

taken four hours after the onset of symptoms showed marked atelectasis involving most of the left lung. Bronchoscopy revealed thick yellow pus oozing from the left main stem bronchus, but no obstruction was noted. Specific antibiotic therapy was immediately instituted. A roentgenogram taken three days later demonstrated considerable clearing. Further recovery was uneventful.

Case vii. A one month old female presented with extensive right sided staphylococcal pneumonia, complicated a few days later by tension pyopneumothorax. Closed and open drainage and specific antibiotic treatment over several weeks improved this to a point where the roentgenologic appearance, except for what we thought was pleural thickening along the lateral chest wall, was satisfactory. However, the infant continued to drain small amounts of purulent material from an open drainage tube and experienced intermittent periods of high temperature. Infusion of dionosil into the right chest showed an unsuspected empyema cavity occupying the upper half and medial base of the right thorax (Fig. 10). Subsequently, the right lung was successfully decorticated.

SUMMARY

The characteristic roentgen changes in primary staphylococcal pneumonia in infants are described and their diagnostic im-

portance in a serious and increasingly common disease stressed. Approximately 85 per cent of 41 cases of proved primary staphylococcal pneumonia in infants seen at The Johns Hopkins Hospital in 1955, 1956 and 1957 exhibited some or all of these changes during the early stage of the disease, permitting etiologic diagnosis roentgenologically, often before it could be made clinically. In about 15 per cent of the infants, the early roentgen manifestations were atypical and roentgenologic diagnosis was not specific. These cases are described in some detail. Neither the clinical nor the roentgenologic course provides adequate explanation for these atypical changes. One case in which continued activity of the disease was suggested clinically, but not roentgenologically, is also described.

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THE VANISHING LUNG TUMOR—PULMONARY HEMATOMA*

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DETERMINATION of the diagnosis of pulmonary tumefaction is an ever present problem. The purpose of this paper is to emphasize the consideration of pulmonary hematomas in a differential diagnosis of the so-called "coin lesions" of the lung.

The formation of pulmonary hematomas by penetrating thoracic wounds is an easily conceivable possibility; however, this potentiality as related to nonpenetrating chest injuries is not as obvious.

During World War II several articles were published on the subject of blast injuries. In 1940, Zuckerman[24] reported experimental results of pulmonary injury to rabbits following blast exposure, demonstrating that the amount of injury was related to the degree of blast and the size of the subject. Also it was found that one side of the animal could be protected superficially with no resultant pathologic changes in the protected lung, whereas the contralateral lung developed hemorrhagic manifestations with microscopic findings of torn alveolar capillaries and interstitial hemorrage. Wilson,[21,22] Savage,[15] and McGrigor and Samuel[8] reported similar changes on postmortem examination of blast injuries as well as disruption of major vessels within the thorax and pulmonary hematoma formation. Because of these experimental and postmortem findings, Zuckerman concluded that the injuries were a result of the direct effect of the blast wave on the thorax. This minimized the importance of the previous theories relating resultant pulmonary changes to sudden distention of the lungs and the so-called suction wave following blast exposure. With these facts, the pulmonary changes subsequent to direct, but nonpenetrating, trauma to the chest become more apparent.

Types of nonpenetrating pulmonary injury due to direct violence with resultant pathologic change include hematoma formation, which is anatomically related to the site of trauma and the contrecoup effect. This latter concept was emphasized by Osborn[9] in 1943, when he reviewed 262 accident cases and observed that a large number of pulmonary changes following nonpenetrating trauma were found localized posteriorly even though the site of trauma was anterior or lateral. Osborn postulated that the thorax is compressed anteriorly and laterally with the posterior portion near the spine being relatively fixed; therefore, the contrecoup exerts its effect posteriorly. Salyer *et al.*[14] in 1953, reported 3 cases of pulmonary hematoma following nonpenetrating chest trauma, one of which showed the hematoma to be on the contralateral side of trauma and rib fracture. Chest roentgenograms revealed single nodular densities localized in the lung parenchyma, and because tumor was considered, operative removal of these lesions resulted. Pathologic examination disclosed findings characteristic of hematoma. In 1950, Welkind[19] reported a case of direct closed trauma to the right chest with the patient complaining of chest pain and hemoptysis for a brief period. The chest roentgenogram demonstrated a spindle shaped density within the lung parenchyma on the side of injury which resolved after a thirteen month period. This density was assumed to represent pulmonary hematoma.

Hematoma formation following wedge or segmental resection of the lung has been observed in about 5 per cent of patients, oc-

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The opinions expressed are those of the author and do not necessarily represent the views of the Navy Department.
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TABLE I

PULMONARY HEMATOMAS

Case No.	Type of Thoracic Injury	No. of Hematomas	Size in Centimeters	Discernible Partial Resolution in Weeks	Complete Resolution in Weeks
I	Nonpenetrating Contrecoup	3	3.5 by 3.5 2 by 1.5 2 by 1	4	16
II	Nonpenetrating Contrecoup	1	3 by 2.3	6	Unknown*
III	Nonpenetrating Direct	1	2 by 2	5	8
IV	Nonpenetrating Direct	1	2 by 1.5	10	16
	Postoperative Segmental Resection Cases	Single	Average 3 to 4	Average 6	Average 20

* Patient discharged before final roentgenogram.

curring within the site of resection. The hematoma becomes apparent only after partial resolution of the pleural parenchymal postoperative changes. Frequently, the potential hematoma presents as a ring-like density circumscribing an area of radiolucency (Fig. 5). A few days later the area will become homogeneous but the central portion may show a relative radiolucency for a week or so. The area then becomes dense throughout and more discrete as contraction occurs. As indicated in Table I, the hematoma will show discernible diminution in size on the average of six weeks and usually disappears after twenty weeks. Exceptionally, these postoperative hematomas have been observed to persist as long as one year.

Roentgenographically pulmonary hematomas may present as single or multiple spherical densities, usually measuring a few centimeters in diameter, or as a spindle shaped density occurring within the lung parenchyma. Changes of pulmonary contusion and rib fractures may be present. The only sign of roentgenographic differentiation from other "coin lesions" is the central area of radiolucency which, at times, occurs within the hematoma, as noted in Case II and the postoperative hematomas. This sign is observed in the early stage of the formation of the hematoma. Later, there appear to be no differentiating factors from other pulmonary tumefactions; therefore, the history is of prime importance.

REPORT OF CASES

CASE I. This twenty-five year old white male was involved in a head-on automobile collision on October 1, 1956. Following this accident the patient was unconscious and several lacerations were noted on the face and extremities at hospital admission. An ecchymotic area was found over the right anterior chest wall with respirations being rapid and shallow. The chest was clear to auscultation and percussion. After regaining consciousness, the patient's chief complaint was right chest pain with accentuation on deep inspiration. This complaint was associated with low grade fever and hemoptysis, both of which subsided following a short course of antibiotic therapy. Roentgenologic study revealed the skull and extremities to be negative. Examination of the chest (Fig. 1) showed fracture of the right first anterior rib with no separation of the fracture fragments. The left chest

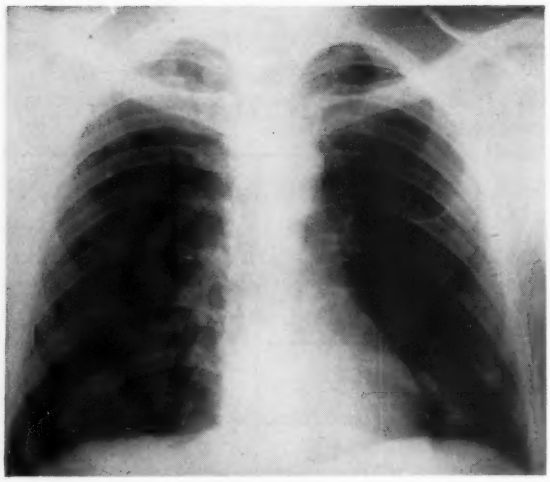

FIG. 1. Case I. Demonstration of 3 hematomas in the left lung field.

revealed 3 discrete nodular densities within the lower lobe posteriorly. The densities measured 3.5 by 3.5, 2.0 by 1.5, and 2.0 by 1.0 cm. The remainder of the examination was within normal limits. Laboratory findings were unremarkable except for a transient microscopic hematuria.

The remaining hospital course was uneventful with the chest pain gradually subsiding. A chest roentgenogram on January 23, 1957 showed complete resolution of the hematomas, four months following the accident.

CASE II. This twenty-nine year old Negro male was hospitalized following an automobile accident with complaints of right anterior chest pain and hemoptysis. Past history and review of systems were noncontributory. Physical examination disclosed a hematoma and abrasion of the forehead. The chest revealed tenderness on the right, anteriorly and laterally, with splinting to the right. Dullness in the right mid posterior chest was noted with a decrease in breath sounds. A few rhonchi were heard in the right anterior chest. Routine laboratory work-up was within normal limits.

Admission chest roentgenogram (Fig. 2*A*) demonstrated an infiltrative process in the posterior portion of the right lower lobe. Fluid was noted in the right lateral sulcus and there was a fracture of the right fifth anterior rib. Serial roentgenograms revealed slow resolution of the pulmonary changes, with a relative radiolucency in the central portion (Fig. 2*B*). One month after admission a homogeneous discrete ovoid density measuring 3 by 2.3 cm. remained (Fig. 2*C*). The lesion was noted to be definitely diminished six weeks after the accident. The patient was discharged asymptomatic.

CASE III. This twenty-two year old white male was hospitalized following injury caused by a discharge of a five inch gun. His complaints were right upper chest pain, hearing difficulty, and hemoptysis.

Physical examination revealed a conscious, apprehensive white male in minimal dyspnea. A laceration of the right infraclavicular area was noted. Bilateral rhonchi and rales were found on examination of the chest. Second degree burns of the neck and head were present. Perforation of the right ear drum was noted. A chest roentgenogram (Fig. 3*A*) demonstrated a massive infiltrative process of the right upper lobe, compatible with contusive changes.

The patient continued to have hemoptysis for about twelve hours. Progressive clinical improvement was noted and on the fifth post-traumatic day the thoracic wound was explored and cardboard wadding was removed. The wound did not penetrate the chest wall.

Roentgenographically, the contusive changes within the right upper lobe showed progressive clearing with the exception of a persistent nodular density (Fig. 3*B*), compatible with hematoma measuring 2 cm. at the level of the second anterior rib. This density underwent slow resolution and eight weeks following injury disappeared completely. The patient was discharged asymptomatic.

CASE IV. This nineteen year old white male was admitted to the Great Lakes Naval Hospital following an automobile accident with findings of a brain concussion, contusion of the left leg, and fractures of the third and eighth left posterior ribs. No other findings on physical examination or roentgenographic study of the chest were noted on the patient's record.

On admission to this hospital, one month following the accident, the patient complained only of pain in the left ankle during exercise. Past history and review of systems were within normal limits except for minimal weight loss noted by the patient before the accident occurred. Positive physical findings were limited to edema, ecchymosis and tenderness of the left ankle. A slight kyphosis was noted. Chest roentgenograms (Fig. 4) disclosed a discrete 2 cm. spheroid density in the lower left lung posteriorly, corresponding to the subapical segment. Healing fractures of the third and eighth

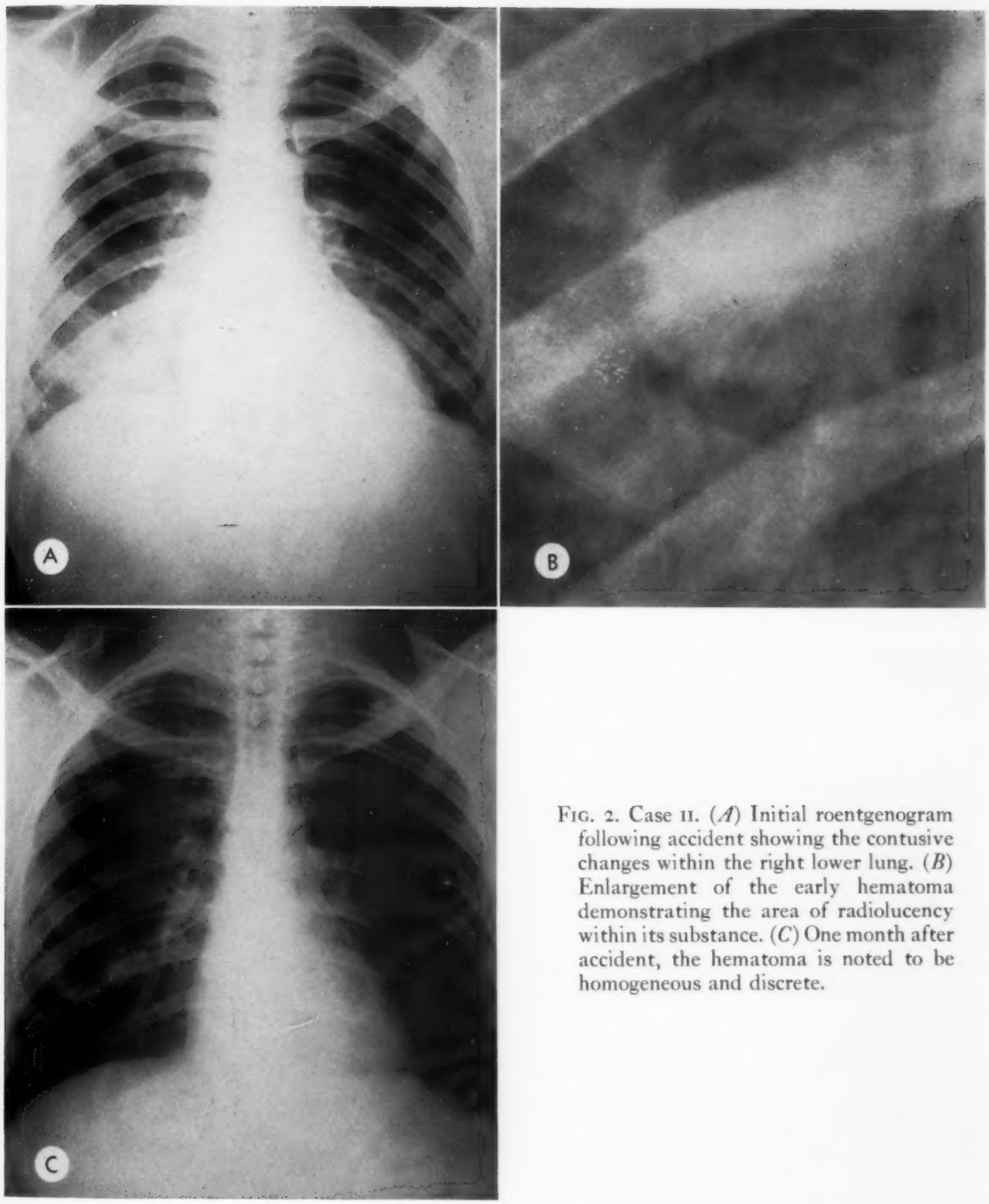

Fig. 2. Case 11. (*A*) Initial roentgenogram following accident showing the contusive changes within the right lower lung. (*B*) Enlargement of the early hematoma demonstrating the area of radiolucency within its substance. (*C*) One month after accident, the hematoma is noted to be homogeneous and discrete.

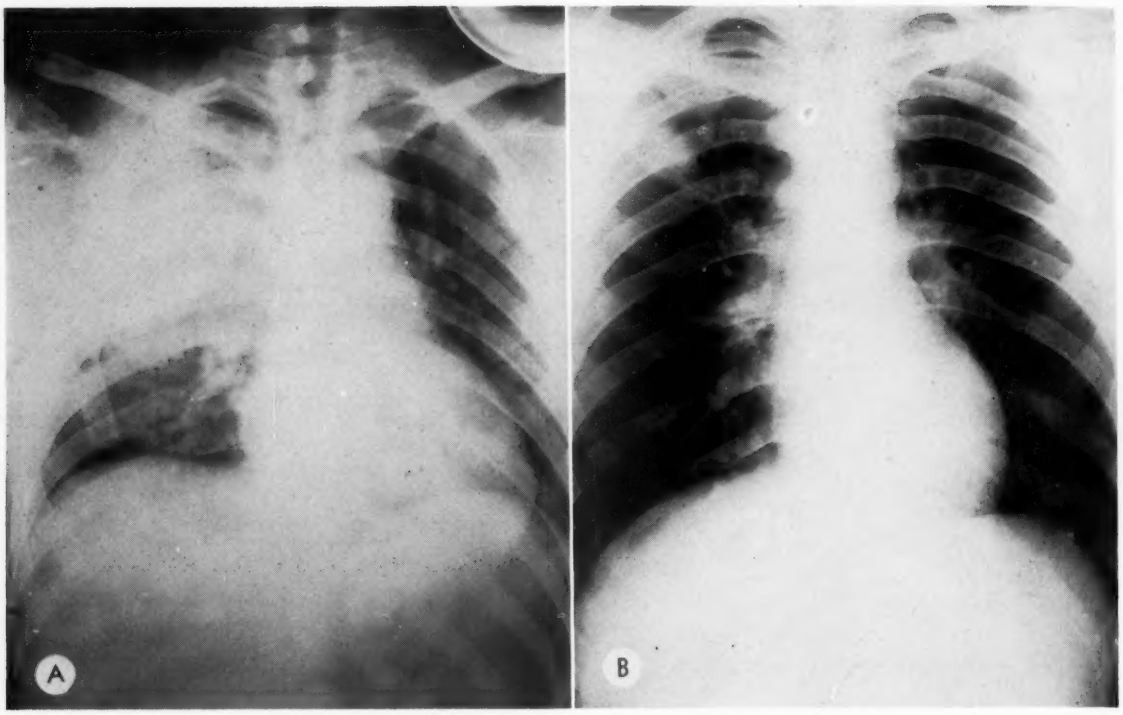

FIG. 3. Case III. (*A*) Roentgenogram showing massive contusive changes in the right chest. (*B*) The hematoma is noted to persist following resolution of the initial contusive changes.

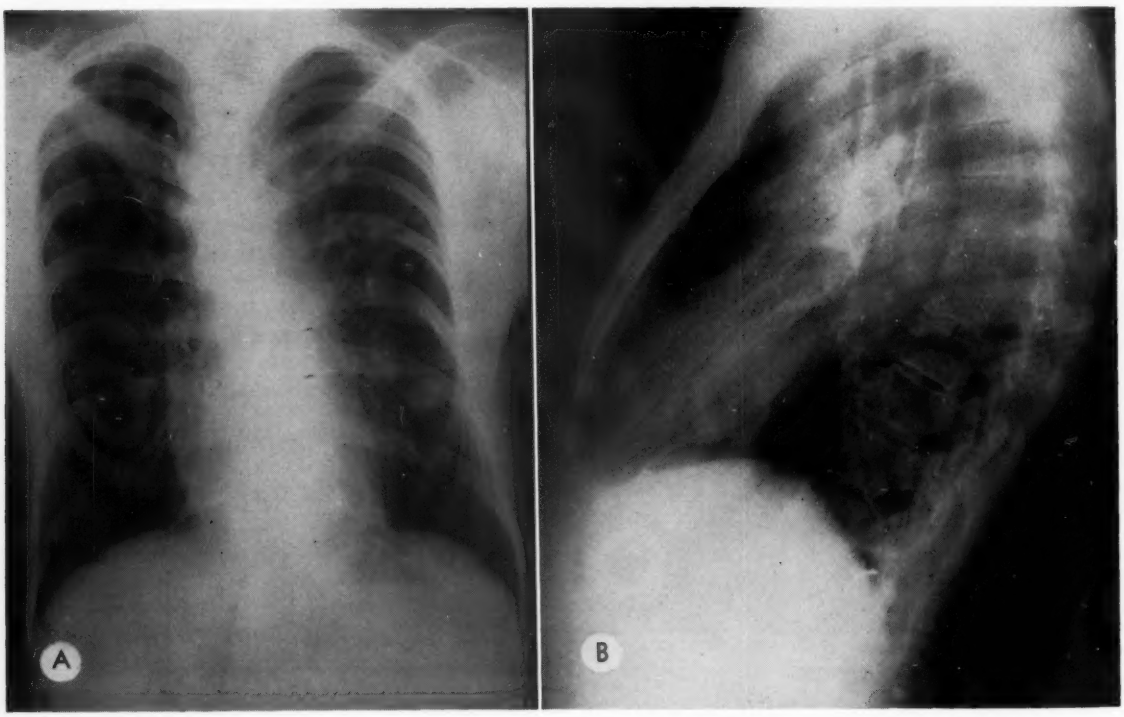

FIG. 4. Case IV. (*A*) Posteroanterior and (*B*) lateral roentgenograms localizing the hematoma to the left lower subapical segment.

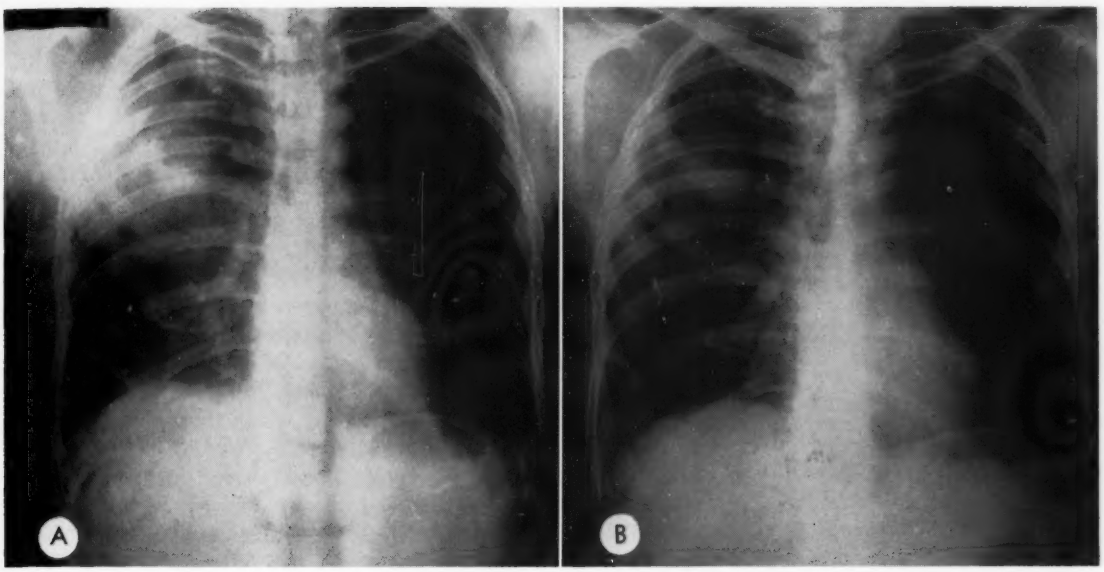

FIG. 5. (*A*) Postoperative segmental resection showing spherical area of radiolucency in which the hematoma is formed (*B*).

left posterior ribs were noted. A kyphoscoliosis was seen. The impression was that the patient had a pulmonary hematoma, with the possibility of granuloma and neoplasm to be considered. Laminagraphic studies revealed the density to be discrete and homogeneous without evidence of calcification. The lesion completely disappeared four months following the accident.

SUMMARY

The etiologic factors and roentgenographic findings in the formation of pulmonary hematomas have been described. Four cases of hematomas following nonpenetrating chest trauma have been presented and the differentiation of these tumefactions from "coin lesions" has been discussed.

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ADDENDUM

A single case of pulmonary hematoma was recently reported by Buechner and Thompson (*Diseases of Chest*, 1958, *34*, 47–54. The hematoma presented as a "coin lesion" within the posterior aspect of the right lower lobe. A history of trauma to the right chest two months prior to the roentgenographic finding was noted. Surgical removal revealed histologic findings typical of hematoma.

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CYSTIC LUNG LESIONS FROM METASTATIC SARCOMA*

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As thin-walled cystic lesions of the lung are rarely associated with metastatic tumor, the following case is being reported. We have not found a similar case recorded in recent medical literature.

REPORT OF A CASE

A twenty year old white male airman had an above-knee amputation of the left lower extremity in November, 1956. Roentgenographically and pathologically the lesion was an osteogenic sarcoma of the distal femur. A preoperative roentgenogram of the chest (Fig. 1) was unremarkable except for a calcified right paratracheal lymph node.

Following amputation the patient had gained weight and felt well. However, in January, 1957 he experienced sudden left lower chest pain which was pleuritic in nature. Repeat roentroentgenograms of the chest had not changed in appearance. The chest pain persisted intermittently and in February, 1957 several thin-walled cystic lesions, some demonstrating fluid levels, were present in both lower lobes (Fig. 2).

The patient began a febrile course in February with temperature spikes to 102°F. daily. Since this hospital is located in a coccidioidomycosis endemic area and serves as a pulmonary disease center, infectious diseases were considered for possible etiology of the pulmonary lesions. Extensive skin tests and repeated complement fixation titers for histoplasmosis, blastomycosis, and coccidioidomycosis were negative. Multiple cultures of the sputum and later of the pleural fluid were all negative for tubercle bacilli, pathogenic fungi and bacteria.

In April, 1957 a roentgenographic examination of the chest (Fig. 3) revealed new cystic lesions with infiltration surrounding most of the lesions. In the upper right hilus and in the left retrocardiac area, solid metastatic lesions were readily apparent. The right hilar lesion underwent cavitation and contained fluid.

The patient's condition rapidly deteriorated. Frequent thoracenteses were necessary for removal of massive hemorrhagic effusions. Four days prior to death, a roentgenogram of the chest (Fig. 4) revealed extensive pulmonary involvement. The patient expired in May, 1957 at another hospital. No autopsy was obtained.

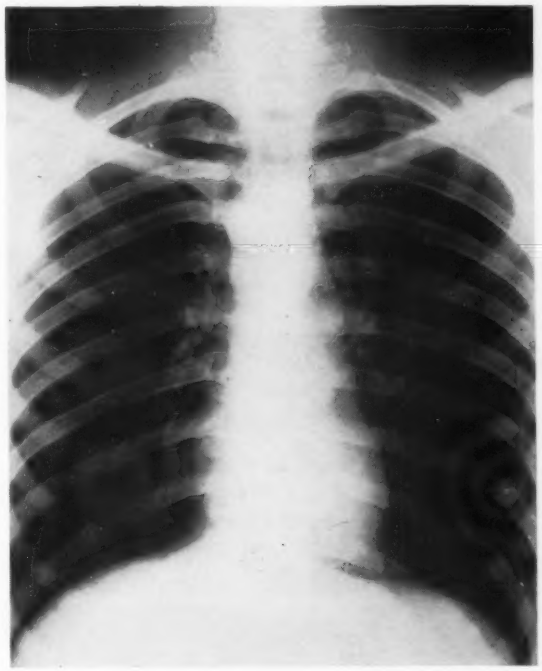

FIG. 1. Preoperative chest roentgenogram. Calicified paratracheal lymph node on the right.

DISCUSSION

This case is of interest due to the initial cystic manifestation of pulmonary metastases. Subsequently, apparent tumor developed. The more common cycle of tumor, necrosis, and then cavity formation did not become evident until late in the course of the disease.

It is believed that this case may further explain the 5 reported cases of "spontaneous" pneumothorax associated with metastatic sarcoma.[1,2] All 5 cases had large tumor nodules when the pneumothorax was diagnosed. Only 2 of the cases had a demonstrable bronchopleural fistula. Lodmell and Capps[1] described subpleural blebs

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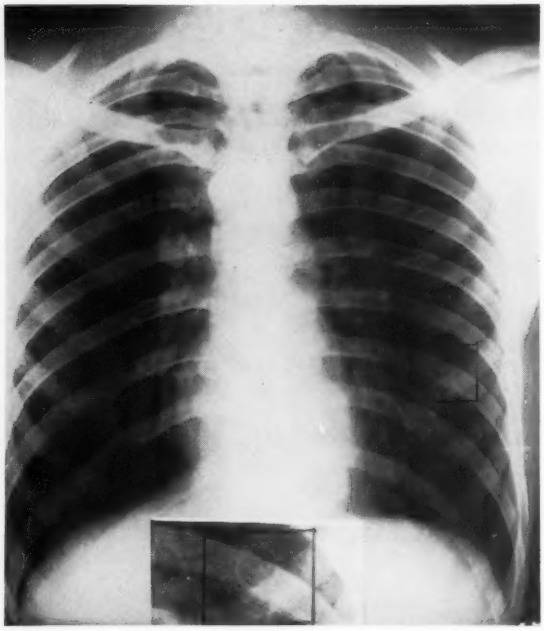

FIG. 2. February, 1957. Roentgenogram of the chest four months after amputation of the left lower extremity for osteogenic sarcoma of the distal femur. Note air-fluid level in cyst below the area shown in the bottom insert.

FIG. 3. April, 1957. Six month postoperative roentgenogram. Large solid metastatic lesions now apparent with bilateral pleural fluid. Note air-fluid level in right hilar mass.

in 2 cases at autopsy. Rupture of these blebs was thought to be the main cause of pneumothorax.

We believe that the manifestation of

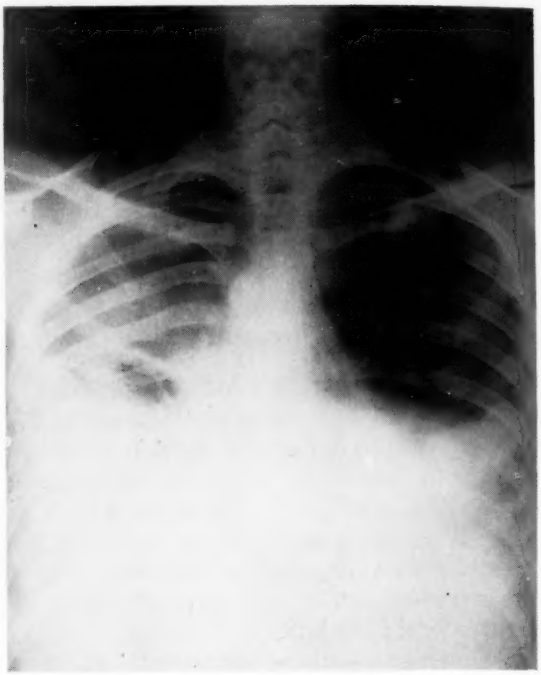

FIG. 4. May, 1957. Chest rentgenogram taken four days prior to death.

cysts in this case prior to roentgen-apparent tumor is an extremely rare occurrence. Tumor necrosis was not a factor in producing the early cysts. This case further supports the contention of Lodmell and Capps[1] that rupture of such tumor-produced cysts or blebs causes the rare pneumothorax seen with metastatic sarcoma.

SUMMARY

A case of metastatic sarcoma to the lungs presenting as thin-walled cysts is reported. The probable relationship of this case to 5 cases of pneumothorax resulting from metastatic sarcoma to the lungs, as found described in the literature, is discussed.

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CRICOTHYROID BRONCHOGRAPHY WITH A POLYETHYLENE CATHETER*

DESCRIPTION OF A NEW TECHNIQUE

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BRONCHOGRAPHY by direct needle puncture of the cricothyroid membrane offers several advantages over the conventional methods of tracheal intubation. Because less local anesthesia is used, it is safer. Because coughing usually is less, filling of the bronchial tree is more complete and the roentgenograms are of better quality. Because the procedure can be performed more quickly and easily, there is less discomfort to the patient.

There are, however, certain undesirable features of the method which have limited its usefulness in bronchography. The head and neck must be kept hyperextended throughout the examination or else the needle is likely to slip out of the trachea. This position makes it difficult to rotate the patient into the oblique positions, often necessary to fill the anterior segments. If the head of the table must be tilted down to fill the apices of the upper lobes, the opaque material may flow into the unanesthetized pharynx and stimulate coughing. The up and down motion of the larynx when the patient swallows may cause the needle to slip out of the trachea. Extravasation of the opaque material into the soft tissues of the neck is difficult to avoid. Repeated reinsertion of the needle into the larynx is likely to cause coughing that may make the bronchogram valueless. In our experience with the method as usually performed, the results were, in general, unsatisfactory due to these difficulties.

There is, however, a need for an alternative method of bronchography when tracheal intubation is difficult or impossible and the several advantages of the cricothyroid procedure by direct needle puncture seem worth preserving. These considerations led to the development of a modification of the technique which eliminates most of the disadvantages of the method while preserving its advantages. A small polyethylene catheter is inserted into the trachea through the needle, then the needle is immediately removed. Bronchograms can thus be taken in any position desired. There is no discomfort to the patient from the tube.

Bronchography has been performed by this method in 25 patients in the Departments of Radiology of The Johns Hopkins Hospital and Union Memorial Hospital. The results have been excellent and there have been no complications. It has proved to be a valuable adjunct to the other methods of bronchography.

REVIEW OF THE LITERATURE

The widespread use of the cricothyroid puncture bronchography with both straight and curved needles, and the relatively few complications reported, attest to the safety of the method. The technique dates back to the original bronchography with lipiodol by Sicard and Forestier[11] in 1922. Since that time this procedure has been employed with increasing frequency, particularly in France and England,[12] and to a lesser extent in this country.[3] It has been used with satisfactory results in a wide variety of pulmonary diseases including active tuberculosis,[2,4] and in all age groups including young children and debilitated elderly people.[1]

Complications reported have been, for the most part, isolated. Köhler[8] reported a case of fatal mediastinal emphysema and air embolism after cricothyroid puncture in a boy aged nine. Olmer and Zuccoli[10] observed transitory air embolism in a man aged thirty-nine. Leroux and Bouchet[9]

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described a case of fatal anaerobic cellulitis. Guy and Elder[6] experienced a breaking off of the needle in the trachea and advised the use of a strong needle. They did not, however, indicate the size or composition of the needle that broke off. Other complications mentioned include hemoptysis, neck pain, hoarseness, dysphonia, extravasation of opaque material into the soft tissues and abscess formation.[6]

Little in the way of statistical evaluation of the complications and reactions has been reported. The implication in most instances is that complications are not common. Guy and Elder[6] said, "Complications are not likely if reasonable precautions are taken." Christopherson[5] expressed the opinion that "the few accidents which occur are due to failure in technique." Burrell and Melville[4] observed no "immediate or late ill effects" in over 100 cases. Amberson and Riggins[1] found one abscess formation in six years' experience in children. The number of cases was not given. In one of the most recent reports (1958) Beck and Hobbs[3] stated that they have used the cricothyroid puncture technique almost to the exclusion of other methods for "many years" with "good results." No complications were noted.

DESCRIPTION OF POLYETHYLENE CATHETER METHOD

The polyethylene catheter method will be described in some detail, since certain pitfalls must be avoided to assure success. The necessary equipment is illustrated in Figure 1.

The patient is placed supine on the roentgenographic table which is then elevated to 40 degrees. A foam rubber pad 6 inches thick and 12 inches wide is placed under the shoulders and the head is allowed to rest on the table or on a small pillow. The neck must be fully extended (Fig. 2). In this position puncture of the cricothyroid notch can easily be accomplished (Fig. 3). Failure to extend the neck fully may result in a failure of the puncture as the needle may slip to the side of the larynx or may puncture the cartilage. The skin over the cricothyroid notch is cleansed with tincture of zephiran in routine manner; then using a No. 22 gauge needle it is infiltrated with xylocaine 1 per cent. Infiltration with small amounts of xylocaine is carried into the subcutaneous tissues and into the cricothyroid membrane. When the tip of the needle enters the lumen of the larynx, 1 or 2 cc. xylocaine is injected. This will stimulate coughing momentarily. If the cartilage has been punctured, usually the lumen of the needle will be occluded by a plug of cartilage. After the anesthetic has been injected into the lumen of the larynx, the needle is withdrawn and a No. 16 gauge thin-walled needle, 1½ inches long, is inserted through the anesthetized area into the lumen of the larynx. Then, with the needle held at a slightly downward angle, a No. PE90 polyethylene catheter is threaded through the needle into the trachea for a distance of about 3 inches. The tube should be threaded far enough so that it will not come out when the needle is withdrawn, and no attempt should be made to withdraw the catheter while the needle is in place as it is possible to cut off the catheter on the sharp edge of the tip of the needle. As soon as the catheter is in place, the needle is withdrawn and the catheter is taped with plastic adhesive tape. Regular adhesive tape is unsatisfactory as it will not adhere to the polyethylene tube. The pad under the shoulders is then moved up under the head as a pillow and taped to the roentgenographic table so that it will not slide when the patient moves (Fig. 2*B*). When the tube first impinges on the wall of the trachea, cough may be stimulated. After injection of another 1 or 2 cc. xylocaine, any sensation associated with the tube ceases. The remainder of the procedure is essentially the same as with other bronchographic techniques.

During the instillation of opaque material through the tube, care must be taken to keep the head elevated above the neck so that the material does not flow into the

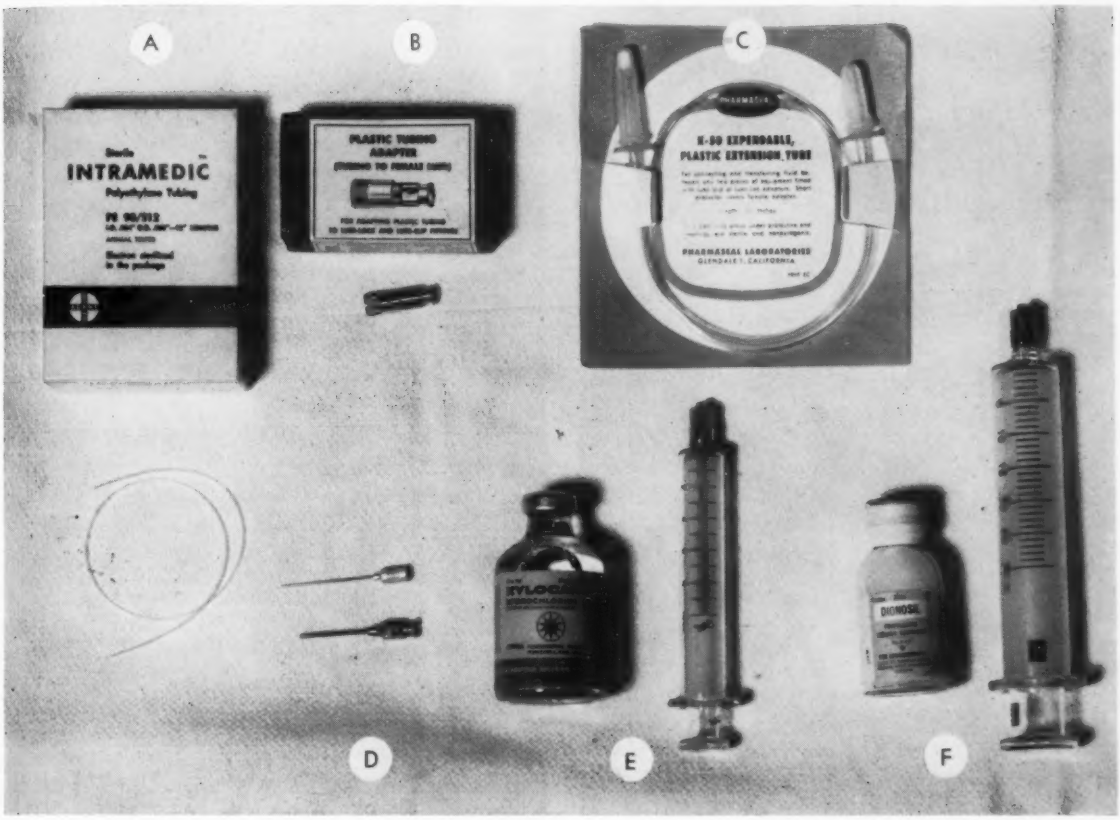

Fig. 1. Equipment necessary for cricothyroid bronchography by means of a polyethylene tube. After the catheter is threaded into the trachea through the needle, the needle is removed. (*A*) The catheter, No. PE90. It is available in bulk form or in sterile 12 inch lengths individually packaged. (*B*) Adapter for adapting the catheter to a syringe or the extension tube. (*C*) Extension tube. It is connected between the catheter and syringe to permit greater freedom of motion of the patient on the roentgenographic table. (*D*) The needles. A No. 22 needle is used for preliminary anesthesia. A No. 16 thin-wall spinal needle cut down to a length of about 1.5 inches is used to thread the catheter (*A*) into the trachea; then the needle is removed. (*E*) The topical anesthetic, xylocaine 1 per cent. Usually 10–15 cc. is used. (*F*) The contrast material, dionosil (aqueous). If visciodol is used, a syringe with control handles is necessary for injection through the small catheter.

unanesthetized larynx. Usually the upper lobes can be filled with the table elevated 20 degrees. It is possible to lower the table to the horizontal position if necessary. If so, another pillow should be placed under the head. A K-50 plastic adapter tube, or similar tube, between the syringe and the intratracheal tube is desirable to allow greater freedom for the operator to position the patient and to minimize the chance of one accidentally pulling the tube out of the trachea.

After completion of the study, the tube is removed without need for subsequent care of the puncture site.

MATERIAL

Bronchography has been performed by this method in 25 cases. Sixteen were in-patients and 9 out-patients. All were adults ranging in age from twenty-six to sixty-eight years. All were followed for periods ranging from a few days to two months. Contrast materials used were dionosil (oily), dionosil (aqueous), lumopaque, and visciodol. A syringe with control handles was necessary to inject the visciodol. Premedication usually was morphine 11 mg. given about one-half hour before the examination. The local anesthetic used was xylocaine 1 per cent. The amount varied

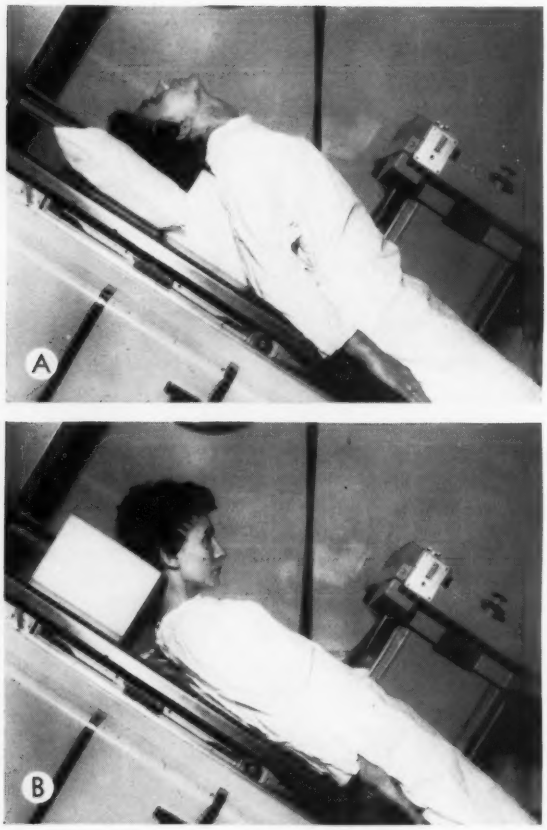

FIG. 2. The position of the patient on the roentgenographic table during the procedure. (*A*) The head of the table is elevated 40 degrees. The cricothyroid notch is placed in proper position for the needle puncture by hyperextension of the head and neck. A plastic foam block 6 inches thick is placed under the shoulders. (*B*) After the polyethylene catheter has been threaded into the trachea through the needle, the needle is removed and the head is placed in flexion on the plastic foam block. It is necessary to keep the head on a pillow so that the opaque material does not flow into the unanesthetized pharynx.

from 8 to 16 cc. Restriction of food and fluid after the procedure was unnecessary since the pharynx was not anesthetized.

RESULTS

Satisfactory bilateral bronchograms were obtained in 24 cases and no serious complications occurred in any case (Fig. 4). In one case, early in the series, coughing prevented filling of the second side after a satisfactory study was obtained of the first side. In this case the coughing appeared to be due to "a tickle in the throat" at the puncture site, rather than to the opaque medium in the bronchi. Almost all patients coughed once or twice at the time of the initial puncture when the anesthetic was injected. Occasionally, there was slight coughing and momentary gagging when the tube was first inserted through the needle. One patient coughed up the tube into the pharynx. Subsequently this was avoided by angling the needle slightly downward while the tube was inserted.

Most patients found the hyperextension of the neck to be uncomfortable enough to volunteer some comment on it. Practically all were apprehensive at the first manipulation of the larynx. One patient fainted for a few minutes at this point in the procedure. Two days later the study was carried

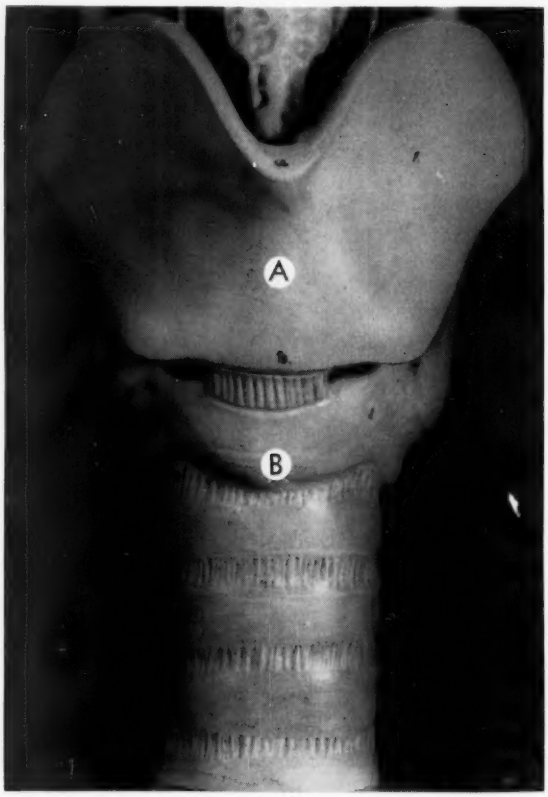

FIG. 3. Model of larynx, external view. The puncture is made through the cricothyroid membrane, just above the superior border of the cricoid cartilage (*B*). This landmark is usually easier to palpate than the inferior border of the the thyroid cartilage (*A*).

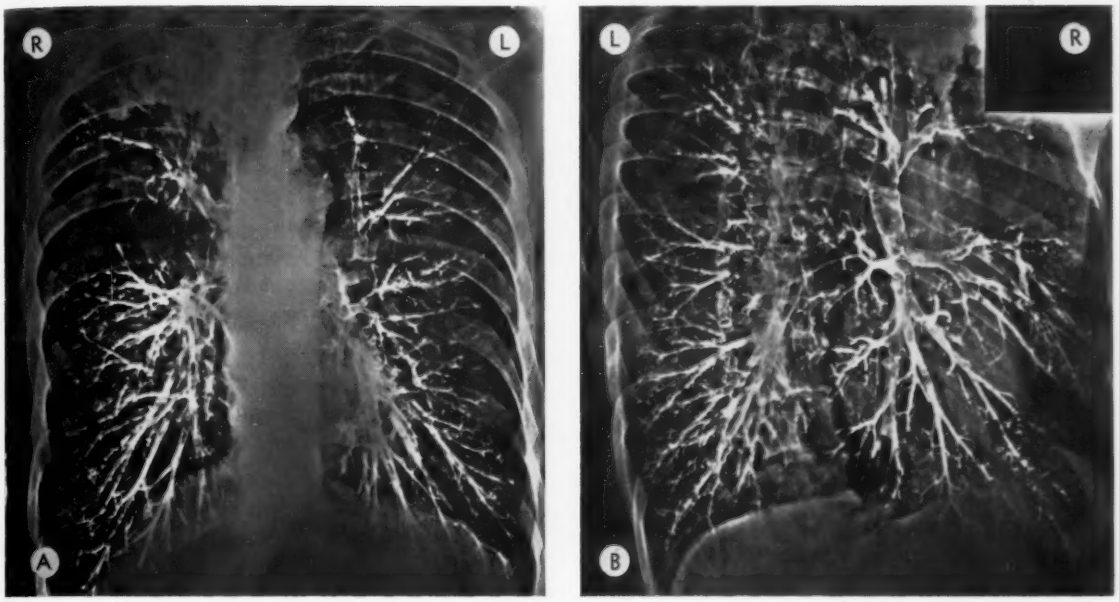

Fig. 4. Bronchogram taken after injection of dionosil (aqueous) through a polyethylene catheter inserted through the cricothyroid notch. Diagnosis: Tuberculosis of the right apex. (*A*) Posteroanterior view. (*B*) Right posterior oblique view (note reversal of this print). Note the filling of the peripheral branches in the anterior portion of the right middle lobe. Complete freedom of motion of the patient permits any position necessary to fill the bronchi in all portions of the lungs.

out uneventfully, but afterward he fainted for a few minutes while he waited to be taken back to his room. Two other patients said they felt faint, but after a moment's wait it was possible to proceed with the study. None of the patients experienced any discomfort associated with the presence of the tube in the trachea. In fact, most said that they could not feel the tube in place. All were able to talk, swallow and move the head without restriction. At the time of the puncture, 4 or 5 drops of blood were noted in most cases. This was of no consequence, and the bleeding stopped almost immediately in all cases. The next day about one-half the patients complained of sore throat when questioned on this point. It was not possible to determine whether this was due to the puncture or to the coughing that usually follows any bronchography. There was no case of dysphonia, hematoma, laryngeal edema, subcutaneous or mediastinal emphysema, local infection or other complication.

Five patients in this series had had previous bronchographies by the nasotracheal catheterization method. Four stated that the cricothyroid method was distinctly less distressing. The fifth patient refused to indicate a choice, expressing equal lack of enthusiasm for both methods.

DISCUSSION

Of the eight different techniques and many variations of bronchography that have been proposed, only the tracheal catheterization methods have gained wide acceptance in this country. While certain workers have become particularly skillful with one or another of the methods that do not require a tracheal catheter, most radiologists and other clinicians prefer the catheter method. This method will probably remain the procedure of choice for most patients. The occasion often arises, however, when there is a need for an alternative method. The cricothyroid puncture with insertion of a polyethylene tube is a safe, simple, easily learned procedure, useful for those patients unsuitable for the catheter method.

In the evaluation of any bronchographic

procedure the major factors to be considered in order of importance are: (1) the hazard to the patient; (2) the quality of the examination; (3) the discomfort to the patient; and (4) the technical skill required to perform the study. A comparison of the catheter method and the modified puncture method, as described above, would serve a useful purpose in establishing on the basis of these factors the choice of the most suitable procedure in any given case.

(1) The relative hazards to the patient are difficult to evaluate because reactions to both methods are uncommon. Few statistical data are available, but it is known that both the catheter method and the direct puncture method with the needle alone have been used for many years in many large series of cases without complications of a serious nature. A number of deaths due to anesthesia have occurred with the catheterization technique, and it would seem that this hazard is greater with this technique than with the puncture method. The anesthetic hazard is increased with the use of the aqueous contrast materials which require more careful anesthetization of the peripheral bronchial tree than is necessary when the oily media are used. Also difficulty in insertion of the catheter may require additional anesthesia and further increase the hazard from this source. On the other hand, a review of the literature shows that complications by the puncture method may occur. In particular, there is the possibility of infection; just as there is with any procedure in which the skin is broken. However, even before the days of modern chemotherapy, infection was observed rarely. Under most circumstances, the puncture method probably is the safer one.

(2) The quality of the examination by the two methods is equally difficult to evaluate. Both are capable of yielding satisfactory bronchograms in most instances. In our experience the problem of coughing has been distinctly greater with the catheter method than with the puncture method, due to irritation of the larynx by the catheter. With the puncture method, the filling of the upper lobes requires special care since it is necessary to maintain the head in an elevated position in order to prevent the opaque material from flowing into the unanesthetized pharynx.

(3) The discomfort to the patient is in most cases less with the puncture method than with the catheter method. This has been the impression of most patients in our series who previously had had catheter bronchography, although it is recognized that insertion of the catheter is a relatively easy procedure. In the present group of patients in whom the puncture method was used, most found the hyperextension of the neck to be uncomfortable and many were quite apprehensive at the first manipulation of the larynx.

(4) The technical skill required to perform the study by the catheter method must undoubtedly be greater than that required for the puncture method. Considerable practice is necessary to master the technique of tracheal catheterization, while the puncture method can be quickly learned. Also, the former method entails more patience and usually the active participation of the patient, while with the puncture method only passive cooperation of the patient is needed.

The factors enumerated indicate that both the tracheal catheterization method and the modified cricothyroid puncture method with insertion of a polyethylene tube are useful procedures, each with its special advantage. Both methods should be available in any clinic where bronchography is done, and the choice of method should be made after evaluation of the individual patient.

SUMMARY

1. A new method of cricothyroid puncture bronchography is described in which a polyethylene catheter is threaded through a previously inserted needle and the needle subsequently removed.

2. The results of 25 bronchographies per-

formed by this method are presented. Excellent technical results were achieved without complication.

3. This method and the tracheal catheterization method are compared. Both methods should be used and the choice should depend on the individual case. It appears that the modified cricothyroid puncture method is preferable to tracheal catheterization when insertion of the catheter may be difficult or may require large amounts of local anesthetics.

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THE AVERAGE DENSITY OF HEALTHY LUNG

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A RELIABLE estimate of the average density of inflated lung is surprisingly difficult to discover. Accurate values would be of some importance in the estimation of dosage in radiation therapy, since the correction for transmission through lung instead of through unit density tissue may be as high as 85 per cent[2,13] and is frequently about 30 per cent.[7] These factors are for 200–250 kv. radiation, but even with Co^{60} radiation the correction may be 20 to 33 per cent.[6] Such divergences from water absorption are obviously much more serious than those due to bone absorption, which do not exceed 2 to 5 per cent in the thorax and 5 to 10 per cent in the abdomen at 250 kv.[7] A critical survey of the data available on lung density, together with information derived by a new method, would appear to be timely.

Spiers[14] in 1951 assumed a lung density of 0.5 gm. per cm.^{3} "in the absence of other information." More recent workers concerned with radiation dosage have quoted values as different as 0.2[12] and 0.5.[10] The present tendency is to accept a value of about 0.3[2] as in the M.R.C. phantom,[16] although a number of comparatively recent references continue to quote higher values. For example, Kornelsen[9] employed a mixture of paraffin wax and sawdust of density 0.46, and Lochman[10] used sponge partially impregnated with water to give a density of 0.5.

It should be appreciated at the outset that as the patient breathes the density of the lungs changes by an average of ± 7 per cent. A more serious problem in radiation dosage is that of congested or growth-filled lungs, where the density may in some cases approach unity. Neither of these sources of error, however, relieves us of the responsibility of determining the best average value for the density of healthy lung. The present paper describes a method which should give reliable densities of human lungs by using a histologic section and integrating the relative volumes of air space and connective tissue.

METHOD

The lungs (from accident cases) were prevented from collapsing by clamping the trachea before opening the chest, and, as nearly as possible in their normally distended state, were fixed and hardened in formalin (formalin 10 per cent, sodium acetate 4 per cent) for three weeks.

Blocks of the fixed and hardened lung ($10 \times 1 \times 0.5$ cm.) after being washed for twenty-four hours in running water were placed in Solution 1 (hematoxylin 5 gm., absolute alcohol 50 ml., distilled water 50 ml.) for thirty minutes, this staining process being carried out in a bell jar under reduced pressure (-40 cm. mercury). They were then transferred to Solution 2 (ferric chloride 10 per cent) for twenty minutes at atmospheric pressure and from this to Solution 3 (iodine 2 gm., potassium iodide 4 gm., distilled water 100 ml.) for fifteen minutes.

After washing for twelve hours, blocks were taken through successive gelatin (10 and 20 per cent) in an incubator at 37° C. and finally embedded in the 20 per cent solution. The gelatin masses were then hardened in 10 per cent formalin for three days.

Sections not more than 12 μ thick were cut on the freezing microtome, mounted on 35 mm. film, rapidly dehydrated in alcohol, cleared with xylol and fixed to the film with a rapidly drying transparent varnish.

The film strips were run through a modified cine-projector so that the images of the

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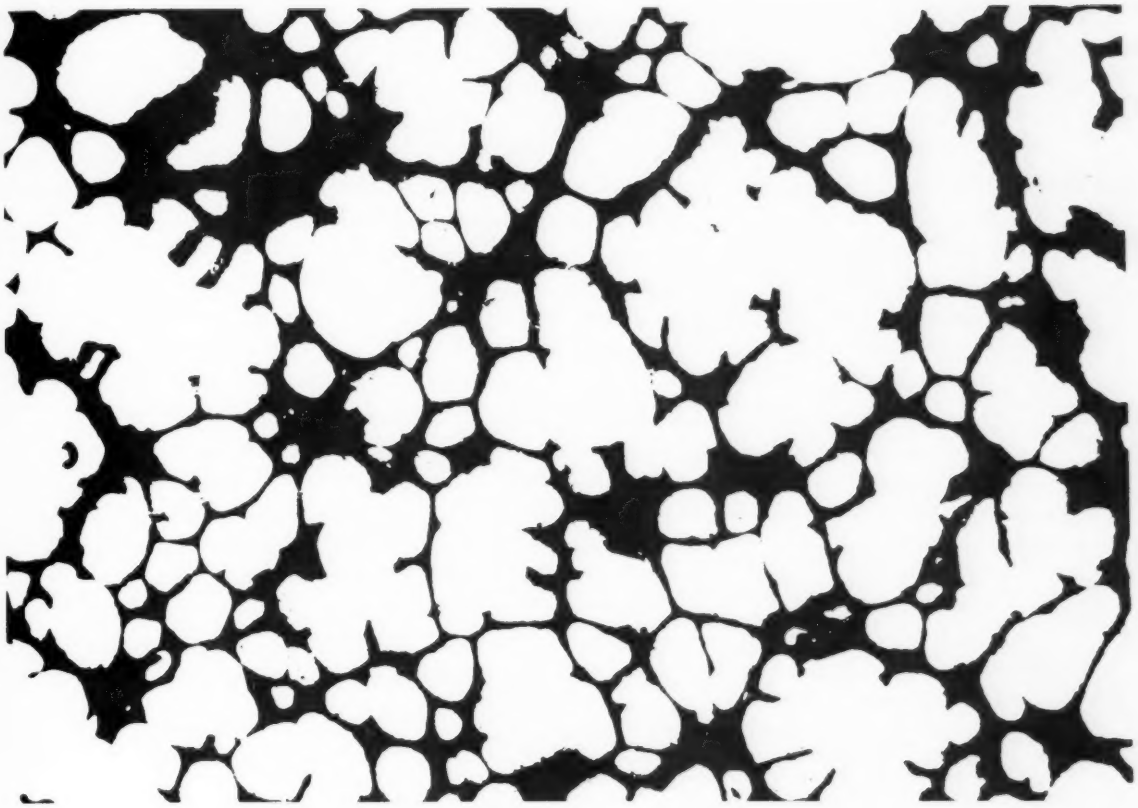

Fig. 1. Lung section 10 microns thick, magnification ×30. When projected onto the screen, the magnification is some 20 times greater.

sections were projected on a screen at a magnification of about ×600 (Fig. 1), the alveolar walls appearing as dark intersections 4 to 15 mm. wide and the alveolar spaces as areas several cm. in width.

The film strip was moved at a uniform slow speed, so that the projected image moved across an aperture in the screen 0.5 mm. in diameter behind which was placed a photomultiplier tube. This tube was arranged to operate two relays, one for "light on" and the other for "light off." Each relay was connected to a simple integrating circuit consisting of an RC arm and a Farmer-Baldwin electrometer. Visual inspection and a manual over-riding control were necessary in order to ensure that large blood vessel sections were counted as "black" even where corpuscles were absent from the section so that it appeared "white" at the center.

An integration of the proportion of "black" to "white" along a narrow strip would give the proportion of unit density tissue to air space along that element of volume. The average density of the lung was given by

$$\rho = \frac{\Sigma L_B}{\Sigma L_B + \Sigma L_w}$$

where L_B is the "black" length, containing the tissue of the alveolar walls and also sections of blood vessels, and L_W is the "white" length, containing air spaces only.

The method was first tested by using a ruler placed across the stationary picture and then by using two stopwatches with the film moving slowly across a point on the screen. Finally, an electrical integration method was adopted to obtain sufficient information to be statistically reliable. Fortunately, the apparatus was already available in almost the form required, hav-

ing been used to obtain estimates of surface area in lungs by counting the number of alveolar walls.[4]

RESULTS

The results on repeated scans over the same track agreed to within ±2 per cent. A number of scans was taken with each section, as shown below. The results on a given lung showed a standard deviation of about 10 per cent, this being due to the different proportions of blood vessels or lung bronchi appearing in the different scans.

Lung section 1: 25 scans	0.240±0.02
Lung section 2: 32 scans	0.205±0.03
Lung section 3: 24 scans	0.265±0.03
Lung section 4: 19 scans	0.316±0.04
Lung section 5: 37 scans	0.272±0.02
Total	0.259±0.033

It is therefore concluded that an average figure for lung density is 0.26 ±0.03.

The method is applicable to a comparison of densities at different anatomical sites in lungs.

DISCUSSION

The main problem is to decide when the specimen lungs are fully inflated, even when this is done *in situ* in the thorax. The lungs used in the experiments above were more likely to be underinflated than overinflated, so that too high densities might be expected. However, a histologic section of lung may give a lower reading of density if most of the blood has drained out of the alveolar walls. These errors tend to cancel each other out and are in any case not expected to be large in the present technique. The results may be compared with the following estimates obtained by other methods.

(*a*) *Anatomic Evidence.* The weight of the average male lungs is given as 1,058 gm. and the collapsed volume as 995 cm.^{3}.[11] This agrees with earlier estimates, for example of Waters,[15] who gave the density of collapsed lungs at 1.062. The average functional air volume in males is 2,920 cm.^{3}.[1] Therefore, the average total volume is 3,915 cm.^{3} so that the density inflated is 0.27, in agreement with the present experimental results (the tidal air is usually taken as 500 cm.^{3}).

(*b*) *Immersion Measurements.* Failla[5] found a density of 0.78 by immersing an inflated lung to determine the volume. The present authors found 0.39, 0.41, 0.81 and 0.44 in four healthy lungs by this technique. The method does not appear to be reliable since air can be lost from the inflated lung by the immersion in water.

(*c*) *Other Estimates.* Jacobson and Knauer[8] froze inflated beef lungs before removing them from the thorax and found densities of 0.43 and 0.32, but used cork of density 0.27 to simulate lung in radiation experiments. Nahon and Hawkes[12] found that a chest phantom consisting of plywood of density 0.5 throughout gave the same radiation transmission as a calf's thorax; the corresponding density of lung would be 0.33 for a 6 cm. wall thickness of unit density tissue and 0.37 for a 5 cm. thickness.

Cohen[2] has proposed using a narrow pencil of roentgen rays to measure the transmission through a thorax, and comparing this with an accurate laminagram to derive the thickness of the lung traversed. Extrapolation from a surgical atlas to give dimensions of the lung section is not a reliable procedure. O'Connor,[13] measuring transit doses and estimating the lung section approximately, found values of 0.3, 0.3, 0.3 and 0.4 in 4 cases for roentgen therapy. More information of this technique is clearly desirable.

CONCLUSION

It appears that values for lung density which were too high have often been used in the past, the correct value being not higher than 0.3 and more probably 0.26 or 0.27. This density was simulated in our phantoms by a mixture of dried coarse sawdust (0.23) and oatmeal (0.72) in sealed cellulose acetate shapes. (The remainder of the phantom was filled with water.)[7,17]

It should be noted that even when a

value for lung density is known, radiation dosage can be calculated at any point in the thoracic section only if an accurate laminagram is available.[6]

The question of lungs containing growth or fluid still confronts the radiotherapist and physicist, and therefore measurements of radiation transmission in individual cases are likely to yield the most relevant information.

SUMMARY

A survey of values quoted by various authors for lung density is given, together with a method of measuring the density by integrating the proportion of air space in a histologic section. This method gives 0.26 ± 0.03 gm./cm.^{3} for the average density of healthy lungs, in agreement with anatomic evidence.

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CERVICOTHORACIC TUMORS: A TECHNICAL AID TO THEIR ROENTGENOLOGIC LOCALIZATION*

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OUR object in this paper is to report and illustrate a simple technique which is a distinct aid in the roentgenography of cervicothoracic tumors. The expression "cervicothoracic tumor" is used here to denote any abnormal soft tissue or bone mass located in the lower neck or upper thorax.

STATEMENT OF PROBLEM

Several factors combine to obscure the definition and localization of soft tissue and other masses in the cervicothoracic region. The difficulty of obtaining uniformly good quality lateral roentgenograms of this area, especially in obese bull-necked subjects, needs no emphasis. However, the technique here described is not intended as a substitute for good quality lateral roentgenograms; it should rather be used to supplement the conventional anteroposterior view.

Roentgenography of soft tissue swellings in the conventional anteroposterior view of the thoracic inlet is rendered difficult by the fact that crossing the field are the osseous clavicles and the flattened first and second ribs which hang fin-like downwards and forwards from the spine; also fanning up and outwards, across the field are the great vascular arches to the head, neck and upper extremities, with their contained blood.

In respect of localization, the chief problem is usually to determine whether or not the mass is intrathoracic, in part or in whole. This will of course depend on its relationship to the plane of the thoracic inlet. This plane joins the upper border of the first dorsal vertebral body, the necks and inner borders of the first ribs, and the jugular notch of the sternum; it slopes obliquely downwards and forwards (Fig. 1). In normal subjects, the horizontal plane through the upper border of the jugular notch of the sternum cuts the vertebral column at a variable level, depending on the age and habitus. This level is usually at the lower part of the second dorsal vertebral body, the disk below it, or the upper part of the third dorsal vertebral body.[1] Thus where it cuts the spine, this plane lies some 2 inches below the posterior part of the thoracic inlet plane.

The obliquity of the plane of the inlet increases greatly with even moderate degrees of kyphosis of the upper dorsal spine. The separation posteriorly between the inlet plane and the horizontal plane through the jugular notch is then correspondingly greater. Thus, in a conventional anteroposterior roentgenogram of a moderately kyphotic subject, a tumor mass 3–4 inches in diameter may, if posteriorly placed (in the paravertebral sulcus), still appear to lie almost wholly above the jugular notch and adjacent first rib, and therefore to be cervical in location.

In Figure 1 are shown the three relevant planes, A–A^1, the plane of the inlet (all tumor mass below this plane is, of course, "intrathoracic"), B–B^1, the horizontal plane through the jugular notch, and C–C^1, the horizontal plane through the junction of the manubrium and body of the sternum; this is the arbitrary lower boundary of the superior mediastinum. The area between A–A^1 and B–B^1 (about one-third of the superior mediastinum) may be "lost" in conventional anteroposterior views being obscured by, and poorly distinguishable from, cervical soft tissues. The lost portion is chiefly posteriorly placed which is also the area most difficult to define in lateral

* Work done under a grant-in-aid from the New South Wales State Cancer Council.
† Working in receipt of a personal grant from The Royal Australasian College of Physicians.

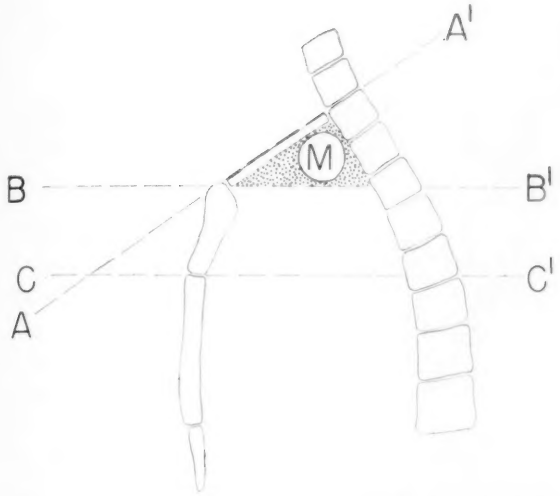

Fig. 1. Diagram showing plane of thoracic inlet (A–A¹), horizontal plane through jugular notch (B–B¹) and lower boundary of superior mediastinum (C–C¹).

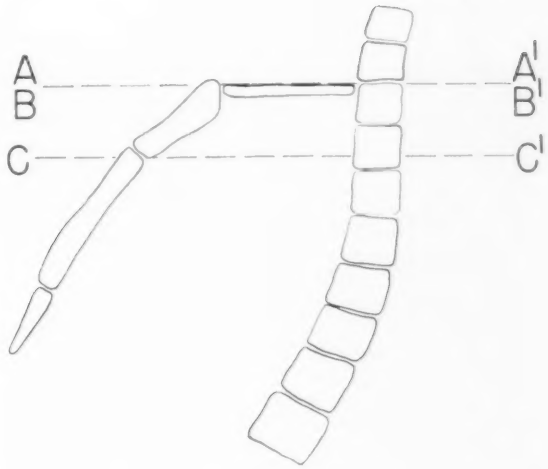

Fig. 2. Diagram showing principle of technique, with plane of thoracic inlet (A–A¹) horizontal.

roentgenograms. The technique here described brings this lost area into view so that soft tissue masses involving it are clearly seen against a background of translucent lung tissue.

TECHNIQUE

Figure 2 illustrates the simple principle on which the technique is based. The body is tilted backwards into a position of increasing lordosis, until the first rib is seen edge on, its anterior and posterior ends lying in the same horizontal plane. In practice we use a 2 mm. focus rotating anode tube. A prominent grease pencil mark is made on the skin just above the vertebra prominens posteriorly and over the upper edge of the jugular notch of the sternum anteriorly. The patient sits with his arm raised above and behind his head, clasping for support a horizontal rod as he tilts backwards until the observer, holding a long wooden ruler in line with the two skin pencil marks, estimates that it is in a plane horizontal to the floor and at right angles to the film (it is convenient to "sight" the ruler to lie parallel with a horizontal line on the wall behind the patient). The tube is centered on the upper edge of the manubrium, the focus-film distance being 6 feet. We use a potential of approximately 73 kv. and an exposure time of 0.07 second at 200 ma., without a Bucky diaphragm. For greater accuracy, immediately before exposing the film, the area may be observed under a fluorescent screen while the patient tilts back into an increasingly lordotic position. When the observer sees the first rib, edge on, in the horizontal plane, the patient is told to hold the position and the film is exposed.

RESULTS

Illustrative Cases. Figures 3 and 4 illustrate conventional and exaggerated lordotic views of a metastatic malignant mass in a forty-nine-year old woman of average build. In the conventional view almost all of the mass appears to lie above the level of the anterior end of the first rib and on this evidence alone its location would, of course, remain uncertain. In the exaggerated lordotic view, however, it is seen to comprise a globular opacity completely within the thorax and its relationship to the left side of the superior mediastinum is clearly defined against a background of lung.

Figures 5 and 6 similarly compare the two views in a patient with intrathoracic goiter. In the conventional roentgenogram the superior mediastinum can be seen en-

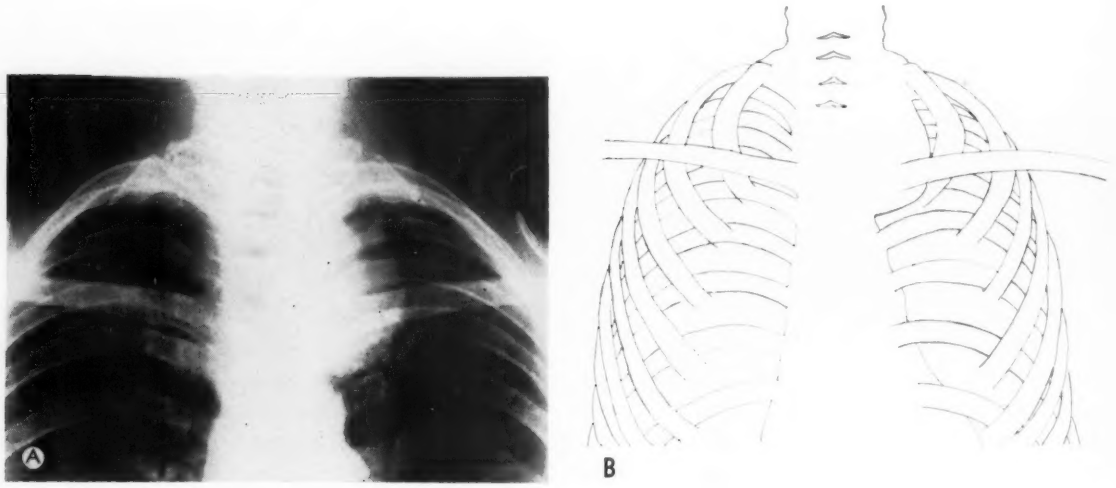

FIG. 3. (*A* and *B*) Conventional roentgenogram and tracing of metastatic malignant mass.

larged to the right but no clear left-sided shadow is demonstrable; yet paradoxically the trachea is displaced to the right. In Figure 6 this tracheal displacement and the explanation of it are clearly shown. A very large left-sided mass is revealed in the superior mediastinum.

At operation it was demonstrated that the right lobe of the thyroid lay mainly in front of the trachea and this was the lobe seen in the conventional view. The left lobe was the larger and, in its descent, it had rotated into the upper posterior part of the superior mediastinum. Though well down into the chest, it thus failed to extend below the horizontal plane of the anterior end of the first rib and therefore did not bulge into the lung fields in the conventional view; nor did it show in the neck, evidently being obscured by the clavicles and vascular arches.

Comment. From Figures 4 and 6 it is clear that the technique described enables the first rib to provide a clear dividing line

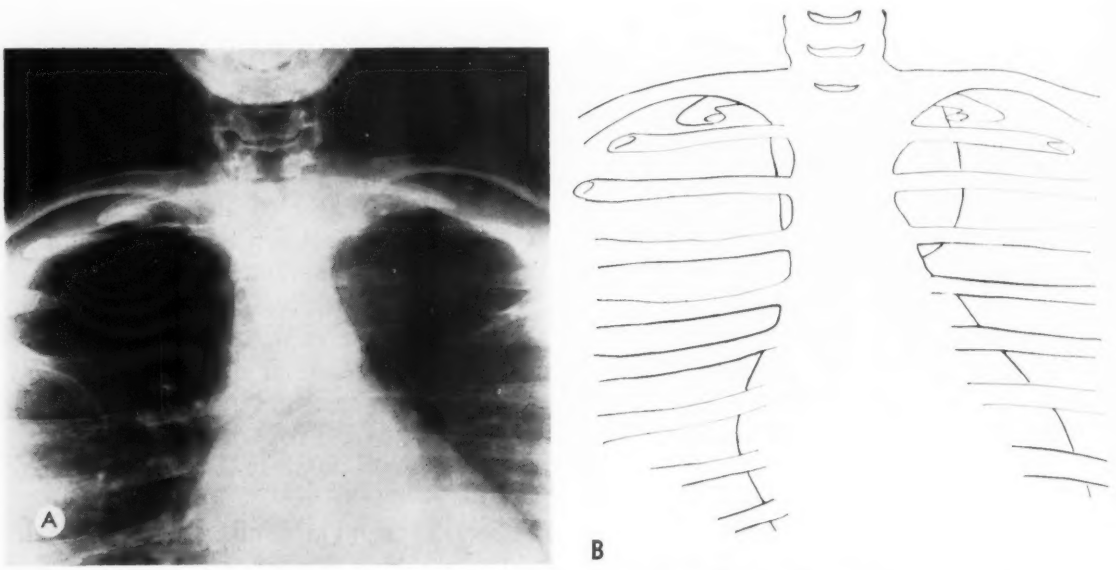

FIG. 4. (*A* and *B*) Roentgenogram in exaggerated lordotic position and tracing of same patient as in Figure 3, showing mass completely within thorax.

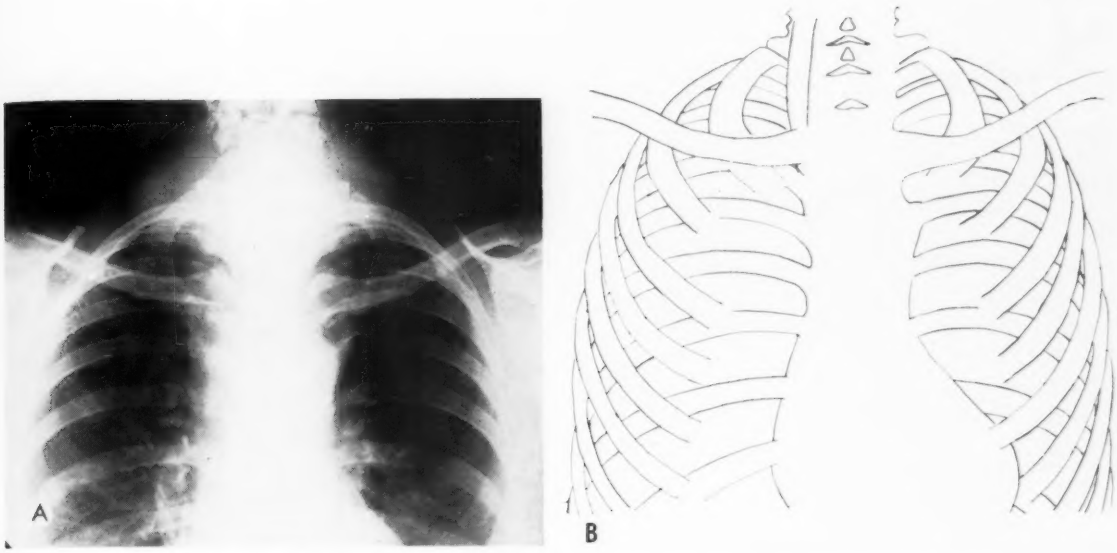

Fig. 5. (*A* and *B*) Conventional roentgenogram and tracing of an intrathoracic goiter.

between the neck and thorax in roentgenograms of this area. Masses in the posterosuperior mediastinum are better visualized than in the conventional view because the roentgenogram is taken below the clavicle and below the vascular arches running up and over the first rib. Moreover, the tumor is seen as through an open, "louvered" window (the ribs being seen edge on) and this is especially well shown in Figure 4.

The technique here suggested was developed to aid in the definition and localization of cervicothoracic tumors. It is significant that a similar but less lordotic posture has long been used as a valuable aid in the visualization of apical lung disease. Also the present technique may help in clarifying the precise site and origin of rib tumors as in the following case: This patient, a male aged sixty-five, presented with a very large, hard, fixed mass in the right infraclavicular region, extending around into the right axilla. A biopsy, done in another hospital, showed it to have the histologic structure of a benign chondroma. Figures 7 and 8 show the conventional

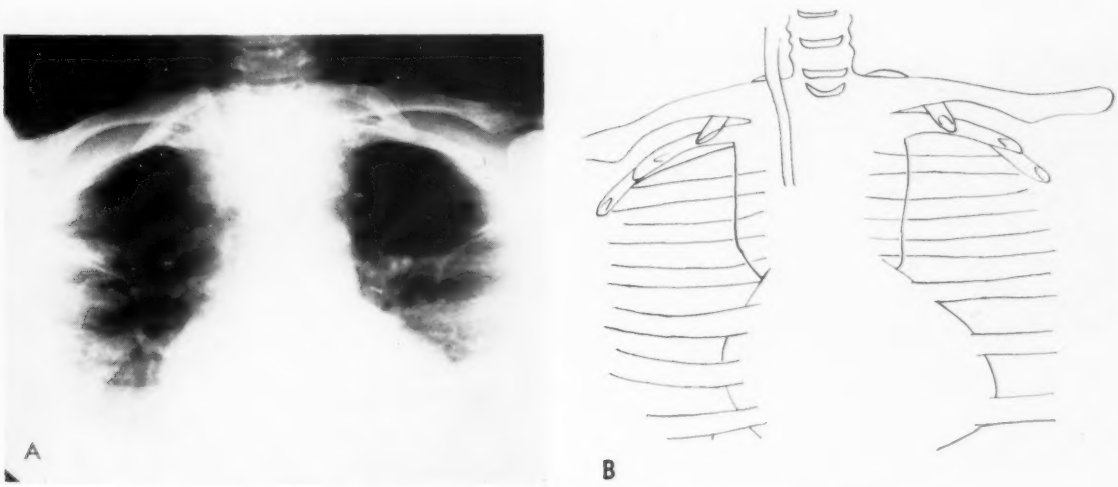

Fig. 6. (*A* and *B*) Roentgenogram in special view and tracing of same patient as in Figure 5, showing large mass in superior mediastinum.

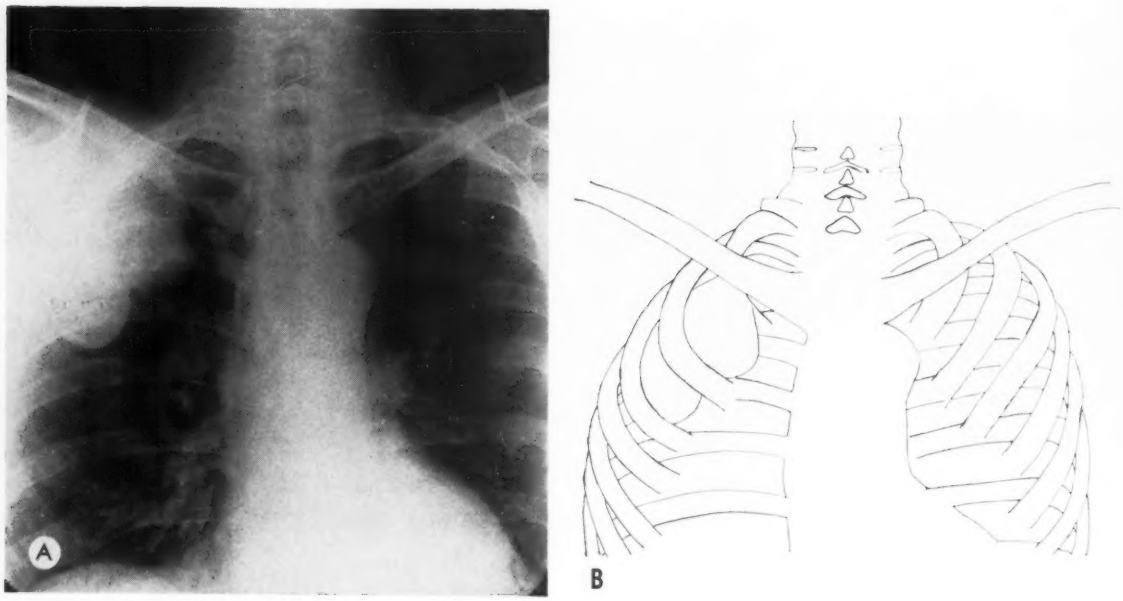

FIG. 7. (*A* and *B*) Conventional anteroposterior roentgenogram and tracing of patient with large fixed tumor in right infraclavicular region.

anteroposterior and exaggerated lordotic views. In the former the tumor is well seen but its relationship to the first, second and third rib cannot be distinguished since their obliquity causes it to overlap all three, but in the exaggerated lordotic position described it is possible to determine that the tumor lies wholly below the first rib and this point would be helpful in planning an operation for its removal.

SUMMARY AND CONCLUSIONS

1. Mediastinal masses protrude into the lung fields which are of a quite different order of density and against which tumor masses are usually clearly outlined. However, because of the obliquity of the thoracic inlet, about one-third of the superior mediastinum, the posterior and upper segment, is overlaid by cervical structures in conventional anteroposterior roentgenograms. Therefore, soft tissue masses in this area tend to be obscured and overlooked. If visualized, it is impossible without other evidence to determine whether they are cervical or thoracic in location. The upper

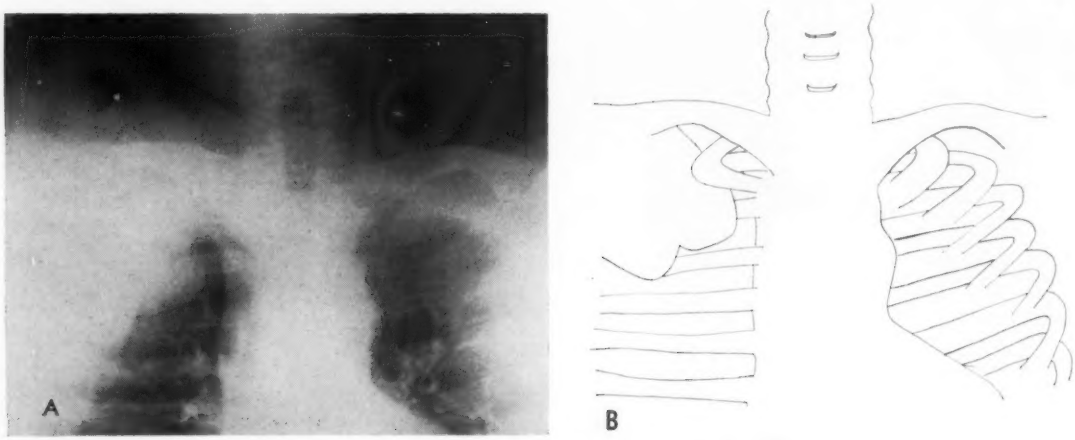

FIG. 8. (*A* and *B*) Roentgenogram in exaggerated lordotic position and tracing of same patient as in Figure 7, showing that the tumor lies wholly below the first rib.

and posterior portion of the superior mediastinum is also the most difficult in which to demonstrate soft tissue tumors clearly in lateral roentgenograms.

2. By tilting the patient into a position of exaggerated lordosis, excellent delineation of mediastinal masses in this obscure area can be obtained against a background of lung. The film is "shot" in a plane below the clavicles and vascular arches of the head, neck and superior extremities, and through the open louvers of the upper ribs which are seen edge on. The simple technique involved is described and illustrated.

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LARYNGOCELE: A CASE REPORT*

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LARYNGOCELE, also known as aerocele, air hernia, laryngocele ventricularis and "monkey's pouch," is a relatively rare abnormality in man with less than 90 cases reported in the literature. By most accepted definitions, this condition may be described as a saccular or cystic dilatation of the ventricular appendage of the larynx which may remain confined within the borders of the larynx (internal laryngocele) or may herniate through the thyrohyoid membrane into the soft tissues of the neck and present as a cervical mass (external laryngocele).

The ventricular appendage in man is generally regarded as an atavistic structure which in lower primates, such as the gorilla, is well developed and probably serves a function in rebreathing. The exact mechanism for the production of abnormal dilatation in man is not certain but it would seem that unusually high intraglottic pressures may explain its formation.

Chronic cough, straining at stool, childbirth, and activities such as blowing wind instruments, lifting heavy weights, and prolonged singing have all been suggested as possible causes. Larrey, a surgeon in Napoleon's army, is credited with describing the first cases of laryngocele in Egyptian priests who served as human loud speakers in chanting the daily verses of the Koran. Also, certain disease processes, especially tuberculosis and carcinoma of the larynx, have been associated with laryngocele formation in a relatively high percentage of reported cases. Inflammatory changes about the neck of the appendage may serve as a check valve mechanism and result in massive dilatation of the structure. Sudden paroxysms of coughing, as in the case reported below, may precipitate an acute dilatation of the sac which may cause severe respiratory distress and even a fatal termination. In most cases, however, the condition is relatively benign.

The roentgenologic findings are fairly typical. Fluoroscopically, it is usually possible to observe the sac moving with motions of the larynx while in cases of external laryngocele with a wide communicating stalk, the air sac may be seen to alternately enlarge with the patient performing the Valsalva maneuver and partially deflate with direct pressure over the cyst. The routine lateral view of the cervical area (Fig. 1) will usually demonstrate the relationships of the air containing sac, depending on its size, but the standard anteroposterior projection is of limited value since detail of the laryngeal soft tissue is obscured by overlying vertebrae and soft tissues. Anteroposterior laminagrams give excellent visualization of the laryngeal structures (Fig. 2). In those rare cases of laryngopyocele, where secondary infection has occurred, a fluid level may sometimes be demonstrated within the sac on anteroposterior and lateral projections taken in the upright position.

Since laryngocele has received relatively little attention in the recent radiologic literature, the following case of an internal laryngocele is considered worthy of report.

REPORT OF A CASE

E. B., a forty-seven year old merchant, was admitted to the Mary Fletcher Hospital because of persistent hoarseness and dysphagia for two days and increasing difficulty with breathing of six hours' duration. The patient stated that he had felt generally well with no respiratory symptoms until three days before admission when he had choked on a lemon peel, this being followed by severe paroxysms of cough-

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Fig. 1. Lateral roentgenogram of the neck shows a large air containing mass occupying most of the larynx and extending up to the level of the hyoid bone. Note the reversed midcervical lordosis.

ing. Physical examination at the time of admission revealed a moderately injected pharyngeal mucosa with tenderness to palpation over and about the region of the larynx; no cervical masses were palpable. There was slight inspiratory stridor noted in the larynx with normal physical findings over the lung fields. The remainder of the examination was noncontributory.

The patient was started on a symptomatic regimen but about ten hours after admission was found in a state of acute partial respiratory obstruction. He was brought to the operating room where a tracheotomy and direct laryngoscopy were performed. At this time a large mass was noted protruding into the larynx on the left side which had displaced the arytenoid cartilage and the epiglottis to the right. The left false cord area was greatly expanded and displaced across the midline making it impossible to visualize the true vocal cords. Aspiration of the mass resulted in a sudden gush of air being released with no evidence of pus noted. Two weeks later the mass was excised at which time a narrow stalk leading into the air sac was found arising from the left laryngeal ventricle. The thyrohyoid membrane was markedly thinned but the air containing cyst had not ruptured through this structure.

Pathologic examination revealed a cystic mass lined by respiratory epithelium consistent with laryngocele.

Fig. 2. Anteroposterior laminagram of the neck demonstrates the marked distortion of the laryngeal structures by the laryngocele. The left false cord is displaced downward and across the midline while the arytenoid cartilage on the left is almost in contact with the opposite side. Note the epiglottis displaced toward the right.

SUMMARY

A case of internal laryngocele is presented. The roentgenologic appearance is typical, particularly when using the laminagraphic technique.

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ADDENDUM

After this paper was accepted for publication, an excellent article entitled "External ventricular laryngocele" by Edward Newell Burke and J. Laurence Golden was published in the Am J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *80*, 49–53.

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TELEVISION CONTRAST EXPANSIONS OF SINGLE ROENTGENOGRAMS*

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VIEWERS of modern television sets know how easy it is to alter brightness and contrast on their monitors merely by a turn of the knob. Settings are easily found which best suit the surround illumination. With the use of a closed television system, it is possible to obtain more contrast enhancement and brightness control in the viewing of a single roentgenogram than can be achieved on ordinary commercial television monitors.[2,3,4]

Our apparatus consists of a closed circuit television system in which the roentgenogram is scanned by means of a flying spot cathode-ray tube and the image is displayed on a monochrome monitor. The speed of scanning is such that a rectangle of light 3×4 inches having 525 lines is formed 30 times a second on the face of the flying spot cathode-ray tube. This rapidly moving spot of light is imaged by means of a lens onto a 6×8 inch area of the roentgen film. The amount of light collected by a photomultiplier tube at progressive instants through each elemental area is inversely proportional to the diffuse optical density of each area. In this manner, a time-varying electrical voltage is generated and processed through special circuits to enhance contrast on a monochrome television monitor.

By clipping steps or ranges of luminance not germane to the area of the film being analyzed and expanding the remaining narrowed portion of the brightness band to the full original dynamic range of contrast, areas 2 per cent or less in contrast difference, areas beyond the range of visual perception, become easily perceptible. The horizontal resolution of the system is approximately twice as great as that of commercial television, not being limited to a 4 megacycle channel. Moreover, the signal to noise ratio also is much higher.

Because radiologists using this system can discern many shadow changes on a monochrome television monitor displaying different aspects of contrast differences not otherwise perceptible, the necessity of obtaining multiple roentgenographic exposures for varying densities is obviated. During actual use of the apparatus, each new setting of the dials brings forth a different level of contrast enhancement so that after viewing the entire progression of levels through the full dynamic range, one is left with the impression not unlike that gained with laminagraphy in spite of the different nature of the information. The contrast enhancement which is achieved occurs with equal ease whether the scanning is done in the darkest or lightest portions of luminance. In other words, over and underexposed portions of the film yield up the maximum of contrasted information when scanned by these television circuits (Fig. 1, 2, 3 and 4).

DISCUSSION

The eye is a remarkable instrument of precision, but has limits when concerned with any method of contrast enhancement of roentgenograms. More than one hundred and fifty years ago, Bouger and later Weber and Fechner observed that when light intensity was gradually increased, the least change in luminance which could be perceived by the eye occurred in a series of steps. In other words, differences in light intensity are perceived in fractions of percentages. The perception of brightness difference between two areas having a luminance of 98 and 100 foot lamberts or 2 per cent will appear to the individual as about

* From the Research Divisions, Department of Radiology, Albert Einstein Medical Center, Northern Division, and Philco Corporation, Philadelphia, Pennsylvania. Read at the Eastern Conference of Radiologists, Philadelphia, Pennsylvania, March 8, 1958.

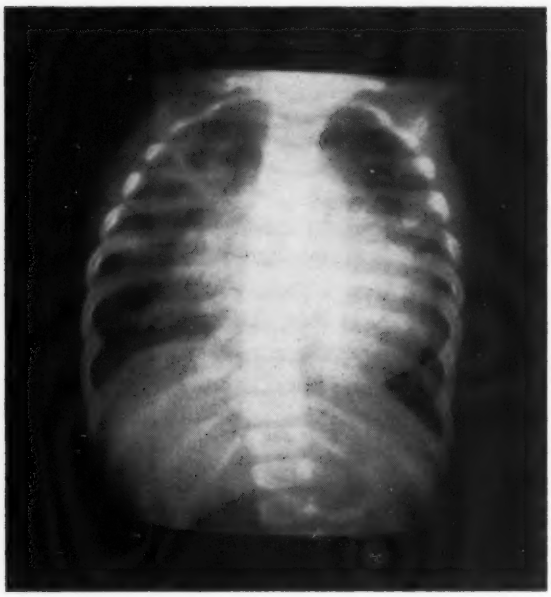

FIG. 1. Chest roentgenogram of infant with disseminated bronchopneumonia as viewed on a television monitor without contrast enhancement.

the same incremental difference in brightness as between two areas of 9.8 and 10 foot

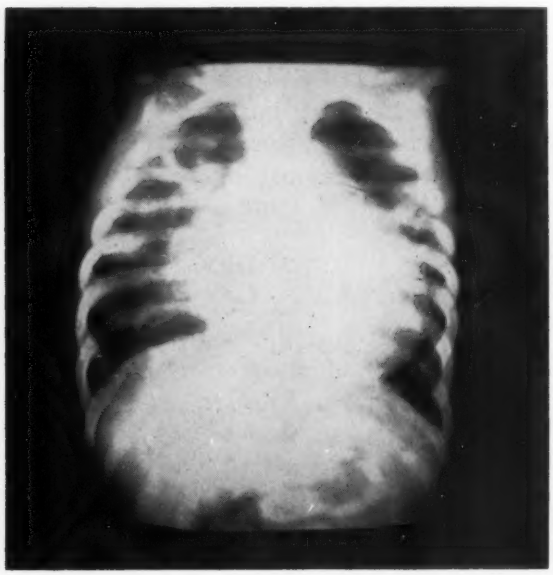

FIG. 2. After a slight turn of two knobs on the control panel, minimal contrast enhancement is gained showing patches of pneumonic infiltrations in both upper lobes more prominently; the differences in aeration of the right and left lungs are accentuated; the right border of the heart is more sharply contrasted; and the ribs stand out in the same type of contrast as would be achieved by a Potter-Bucky examination.

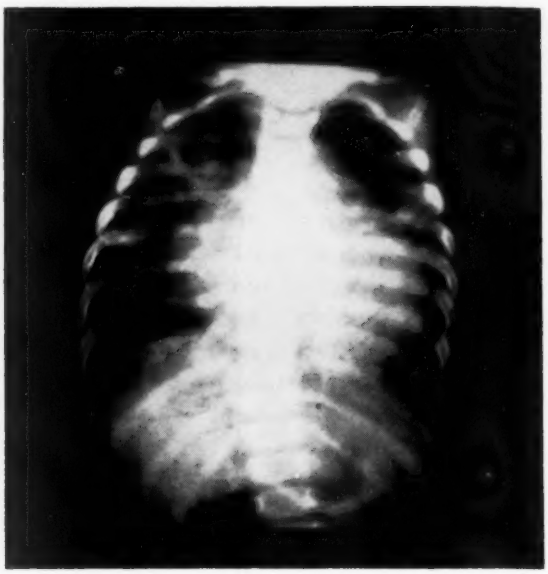

FIG. 3. With a further slight turn of the control television knobs, a Potter-Bucky-like view of the chest is obtained revealing portions of the spine and the ribs over the liver. The hilar and mediastinal structures are now brought into better contrast.

lamberts which is also 2 per cent. This is approximately the lowest fraction of contrast difference which the eye can see in the *photopic* range of the Weber-Fechner curve, a curve which plots the percentage differ-

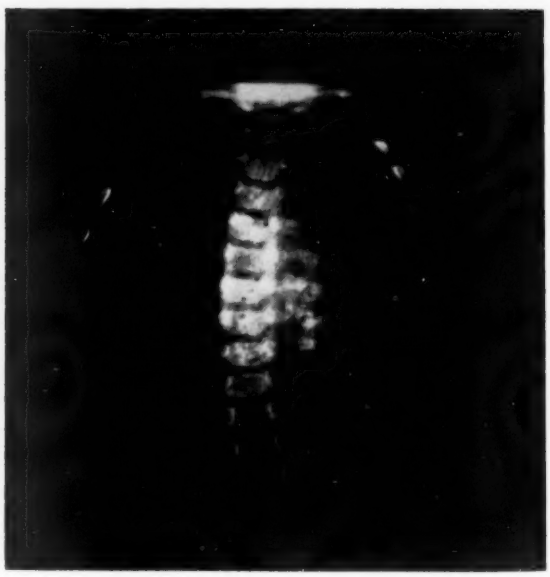

FIG. 4. A further turn of the television control knobs increases the contrast markedly, bringing into view the entire dorsal spine and the left hilar structures behind the heart.

ence of luminance against the log of luminance in foot lamberts. The law breaks down in the scotopic and glare regions; but in reading roentgenograms, only the photopic portion of the curve is pertinent. Data by Blanchard and Lowry as reported by Major Adrian Cornwell-Clyne[1] show that the threshold of contrast perception remains fairly constant, about 2 per cent over a range of brightness from about 0.1 to 150 foot lamberts.

The luminance in reading roentgenograms ranges from a fraction to about 150 foot lamberts so that the 2 per cent limit of contrast perception pertains to our problem. Here is where television techniques can be used to great advantage.

The use of our television system not only makes possible a contrast expansion analysis of a roentgenogram, but it also increases the contrast difference between adjacent areas, where ordinarily the eye would fail to notice the minimum contrast differences. From routine films, conversion to contrast similar to that gained by the use of the Potter-Bucky diaphragm is thus made available to the radiologist without the necessity of multiple exposures to the patient.

SUMMARY

The principles of a television technique have been described for multiple contrast enhancements of various levels of luminance facilitating interpretation of single roentgenograms. Re-examinations or multiple exposures for different contrasts are obviated since enhancement in contrast is obtained with these television techniques over all regions of a single film, whether over or underexposed.

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AN ADAPTOR FOR DEVELOPING 70 MM. ROLL FILM IN THE EASTMAN X-OMAT*

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THE Eastman X-Omat film processor, designed for the automatic processing of cut roentgen-ray film, will handle 70 mm. roll film as well if certain accessories are provided. The required accessories are illustrated in Figures 1 to 6, with the individual parts numbered 1 to 12.

LEADER

It is necessary that a stiff, flat leader (9) be attached to the end of the roll film, a convenient material being a strip of unexposed roentgen-ray film 70 mm. wide and 14 inches long. The technician is instructed that before cutting the exposed film the chest microfilm apparatus is to be cycled fifteen times to provide approximately a meter of unexposed film at the end of the run. In the darkroom (Fig. 2) the cover (3) is removed from the magazine (2) by removing the nut (4) and in total darkness the spool of exposed film is inserted in the magazine with a few inches of the unexposed end protruding through the slit in the throat. The cover is now re-attached and the lights turned on.

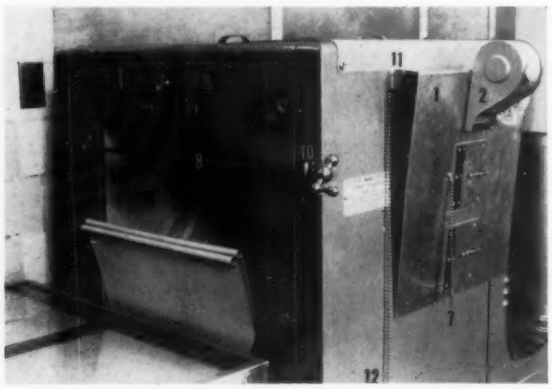

FIG. 1. Outlet of X-Omat, showing skewer and take-up reel in place and adaptor stored on its bracket. (1) Base; (2) magazine; (7) scriber; (8) skewer; (10) skewer bracket; (11) base bracket; (12) scissors (attached to end of chain).

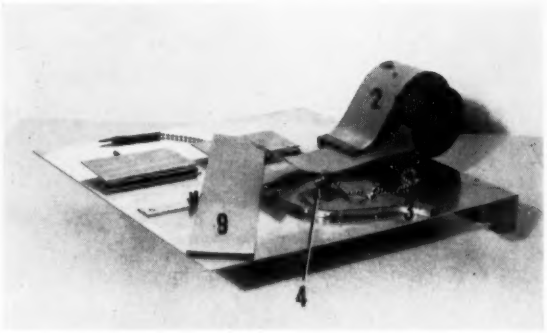

FIG. 2. Magazine cover removed for insertion of roll of exposed film preliminary to attachment of leader. (2) Magazine; (3) magazine cover; (4) magazine nut; (9) leader.

Working in the light, the end of the film is placed in clamp A (5) (Fig. 3) and with the scriber (7) a deep line is scratched across the film, using the distal edge of the gate of clamp A as a straight edge. Now the clamp is opened, the film broken off along the scribed line and then reinserted in clamp A with its end projecting beyond the edge of the gate to line B, which is engraved on the aluminum base (1) $\frac{1}{4}$ of an inch proximal to clamp B (6). A piece of leader is inserted in clamp B with its proximal edge overlapping the end of the roll and coinciding with line A engraved on the base, thus providing a $\frac{5}{8}$-inch overlap of leader on roll film.

MAKING THE SPLICE

Strips of waterproof adhesive plaster, $\frac{1}{2}$ inch wide and 4 inches long, are placed across the overlapped film and leader (Fig. 4). Both clamps are now opened and the ends of the adhesive are stripped away from the aluminum base, taking care not to disturb the relationship between roll film and leader. The four free ends of adhesive

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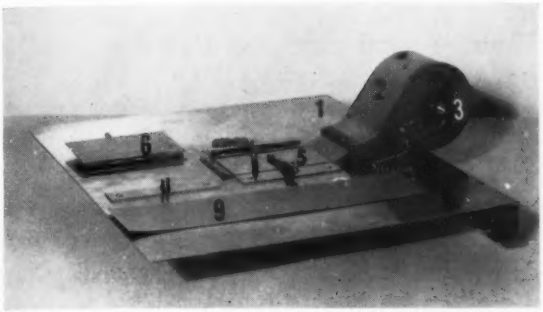

Fig. 3. A roll of exposed film is in the magazine and the cover has been replaced so that work can go on in the light. The end of the exposed film is in clamp A, ready for squaring of the end with the scriber. (1) Base; (2) magazine; (3) magazine cover; (5) clamp A; (6) clamp B; (9) leader.

are folded under and pressed home against the back of the splice and, if desirable, the splice may be further reinforced by stainless steel staples. When the splice has been completed (Fig. 5), the film is shoved back into the magazine and the base is placed on the entrance shelf of the X-Omat (Fig. 6).

PREPARATION OF THE X-OMAT

Eventually it may prove to be worthwhile to slow the speed of progression or increase the temperature of development in order to accomplish more complete development of the single emulsion 70 mm. film, but for the present we are using normal time and temperature and obtaining excellent results.

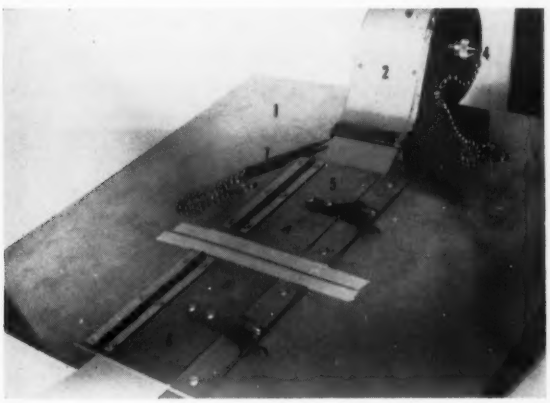

Fig. 4. The leader has been overlapped with the film and clamped with clamp B and adhesive strips are in place as a preliminary step in splicing. (1) Base; (2) magazine; (4) magazine nut; (5) clamp A; (6) clamp B; (7) scriber.

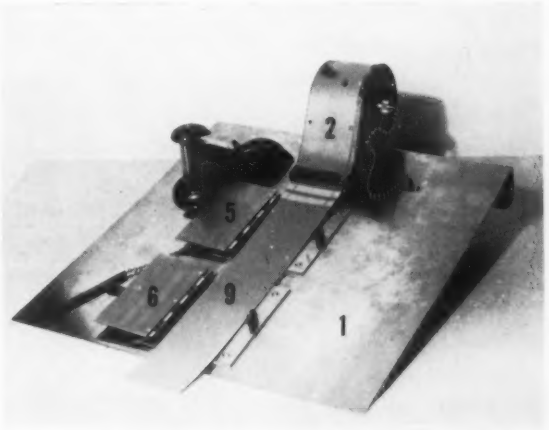

Fig. 5. Adhesive strips have been folded under to complete the splice and in this instance stainless steel staples have been added as well. The film has been shoved back into the magazine and the assembly is ready for placing on the entrance of the X-Omat. (1) Base; (2) magazine; (5) clamp A; (6) clamp B; (9) leader.

The replenisher pumps are turned off, the entrance red light is disconnected, and with the room in complete darkness the end of the leader is inserted in the entrance rollers. As the splice passes these rollers, the reject circuit operates and its signal sounds, but, when the reset button is pushed, the signal ceases and the leader passes on normally through the apparatus, dragging the roll film after it. As processing

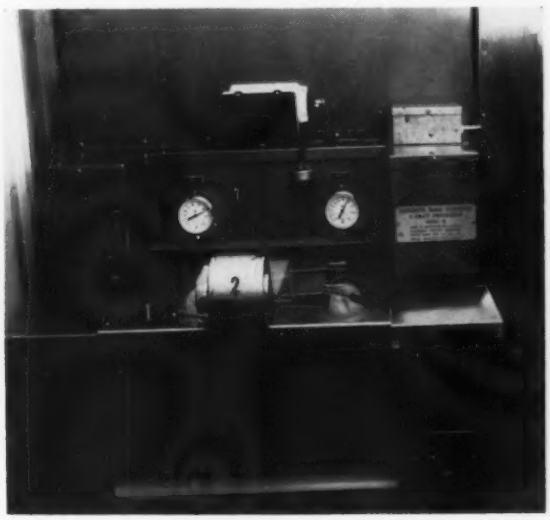

Fig. 6. Adaptor in position on entrance bed of X-Omat; leader ready to be fed into entrance rolls. (1) Base; (2) magazine.

goes on, the technician turns on the replenisher pumps for periods of 25 seconds for every 100 exposures on the roll.

TAKE-UP SPOOL

An empty film spool is slipped over the skewer (8) and the latter is inserted in its brackets (10). One technician remains in the darkroom to operate the replenisher pumps and to resume the processing of cut film as soon as the last of the roll has entered the X-Omat and a second worker is stationed at the outlet. As soon as the leader appears, this latter worker exerts moderate traction on it until the splice is delivered and then with scissors (12) cuts off the film proximal to the splice and discards the leader. The cut end of the roll is now attached quickly to the take-up spool and, by means of the crank at the end of the skewer, a gentle but continuous pull is exerted as the completely processed film is wound onto the take-up spool.

STORAGE OF APPARATUS

The empty spool remains in the magazine to become the take-up spool the next time the apparatus is used. The skewer is hung in a bracket at the side of the X-Omat which is obscured by the base assembly in Figure 1, and the base assembly itself is hung on its brackets (11) (Fig. 1).

SEMI-AUTOMATIC APPARATUS FOR USE WITH CONVENTIONAL DARKROOM TANKS

Those who have had actual experience processing 70 mm. film are well aware of the limitations of conventional methods. In most hospitals the daily grist of 70 mm. chest microfilms can well wait until early evening for processing in the X-Omat to avoid interfering with the more pressing needs for the processing of cut films. Occasionally, however, when large surveys are being conducted on staff, students, etc., it is desirable to be able to process 70 mm. films in conventional tanks. Semi-automatic apparatus for such work is now in production in our laboratory at the University of Chicago and will be published shortly in this JOURNAL.

Arrangements have been made for the production of both devices by a Chicago manufacturer.

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PLASTIC ROENTGEN-RAY PHANTOMS

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REDUCTION of radiation exposure to human beings is of prime concern to everyone associated with the science of radiology. The moral obligation to eliminate all unnecessary exposure is generally recognized. One phase of our approach toward meeting this obligation has achieved a degree of success and forms the subject of this paper.

PROBLEM

Since the discovery of the roentgen ray, demands by radiologists and researchers for human-like test objects have been repeatedly made. Phantoms have been needed to facilitate detailed and accurate experimentation in the study of radiation dosage, as well as evaluations of equipment, accessories, and exposure techniques in roentgenology.

Several approaches have been made toward the development of satisfactory test objects or roentgen-ray phantoms. The work by Harris and co-workers[3] in perfecting their excellent chest phantom is an example. The problems involved concern (1) the availability of components, (2) skilled manpower, and (3) the fragility of the phantom.

A review of the potential uses and the characteristics required of a group of roentgen-ray phantoms indicated that a new attack on the problem could result in obtaining phantoms with a satisfactory combination of properties. The need for phantoms appeared to be greatest for use in comparative evaluations of (1) roentgen-ray exposure techniques, (2) roentgen-ray apparatus and accessories, and (3) roentgen-ray films and fluorescent screens, as well as (4) measurements of radiation dosage. In addition, it was evident that full-scale transparent phantoms containing human bones with normal surface anatomy would be valuable in teaching roentgenographic anatomy, positioning and exposure techniques. It was concluded that the material used to prepare roentgen-ray phantoms should be (1) roentgenographically comparable to human tissue in absorption of primary and emission of secondary radiation, (2) able to penetrate dry bones and replace the air contained therein, (3) safe and reasonably easy to process, (4) stable under normal conditions, (5) durable, and (6) if possible, uncolored and transparent.

A survey of 16 different materials was made with the assistance of N. G. Fisher and L. S. Conley, Du Pont Central Research Laboratory, and F. P. Alles, R. W. Upson and R. Armstrong, Parlin Research Laboratory. At the same time, W. C. Gamble[1] was consulted because of his company's* experience in embedding biologic specimens in a transparent plastic called Bio-Plastic. Roentgenographic evaluations were made of 4 candidate materials including Bio-Plastic, using exposure techniques covering the 40 to 130 kv. peak range. Bio-Plastic demonstrated an absorption coefficient and secondary radiation characteristics essentially equal to human tissue. Since this material also met most of the other requirements listed above, further investigation of the suitability of this plastic for roentgen-ray phantoms was conducted.

An elemental and physical comparison of Bio-Plastic to the human body and to phantom materials in current use was made. The results, listed in Table 1, showed the plastic to be more similar to human tissue than some of the other compositions.

Bio-Plastic[1] is a casting plastic of the thermosetting polyester family. Liquid monomer is polymerized—changed to a solid—by the addition of a catalyst. The material can be handled with a minimum

* Ward's Natural Science Establishment, Inc.

TABLE I

BASIC ELEMENTAL AND PHYSICAL COMPARISON

(Approximate values)

Element	Atomic No.	Human Body[2]	Bio-Plastic[1,*]	Paraffin[4]	Pressd-wood[5]	Water
Oxygen	8	66%	24%	—	40%	88%
Carbon	6	17.5%	70%	86%	53%	—
Hydrogen	1	10.5%	6%	14%	7%	12%
Nitrogen	7	2.4%	0.02%	—	—	—
Average Atomic No.		7.00	6.18		6.45	7.16
Specific Gravity		1.02	1.20		1.05	1.00

* Minus bone.

of special apparatus and is comparatively inexpensive.

PROGRAM

The initial effort to embed bones in this plastic was spent on a small-scale phantom. A foot bone was embedded in a 3 cm. layer of plastic. Roentgenographic evaluation of the object revealed that it had absorption characteristics essentially equal to those of a human foot. However, air trapped in the bone cavities caused atypical and objectionable high density shadows. This deficiency was eliminated in a duplicate phantom made under a partial vacuum with air pressure reduced to 200 mm. of mercury.

A survey of available know-how[6] indicated that a reasonable assurance of success could be expected from a program to make a full-size phantom. Therefore, a human lumbar spine and sacrum were embedded in a large block of Bio-Plastic. The object measured 13.5 cm. deep, 34.5 cm. long and 28.5 cm. wide. Casting and curing were done at intervals over a two week period. The result contained some cracks caused by the excessive amount of incidental, internal heat generated during polymerization. Nevertheless, the phantom proved entirely satisfactory for use. A roentgenographic study showed that it was comparable to a human lumbar spine and pelvis in essential characteristics.

In addition to this phantom (Fig. 1), 2

FIG. 1. Photograph showing human lumbar spine and sacrum embedded in Bio-Plastic. Cracks caused by excessive, incidental internal heat generated during polymerization are visible. Lead strip at lower margin on bottom prevents exposure from reaching film while holes drilled in block cast dark shadows.

blocks of plastic were prepared to superimpose on top of the "pelvis" phantom to bring the total thickness to 19.5 cm., or approximately the same thickness as an average human pelvis. Roentgenographic evaluation proved that the phantom was a satisfactory substitute for a human in anteroposterior pelvis and lumbar spine studies (Fig. 2), as well as in lateral views made with the block on its side.

The program was reviewed at this stage

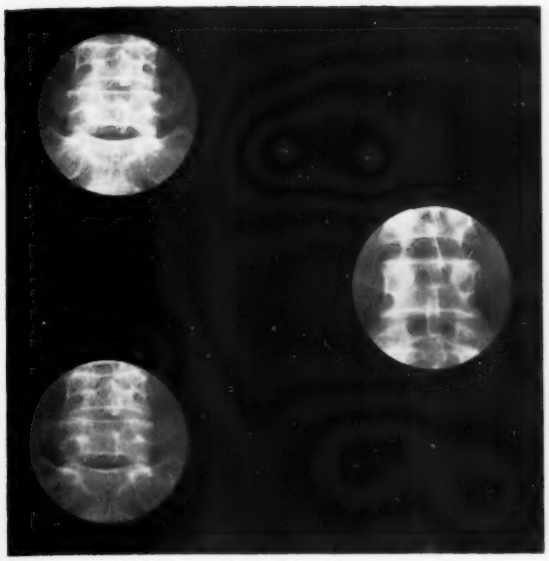

Fig. 2. Reproduction of anteroposterior roentgenograms made using the "spine" phantom (19.5 cm.) and a human spine (23 cm.) at 40 inches with a 16:1 grid. Hi-Speed intensifying screens and "Xtra-Fast" roentgenographic film. *Upper left:* phantom, 70 kv. peak and 90 ma. sec. *Right:* human, 70 kv. peak and 112.5 ma. sec. *Lower left:* phantom, 90 kv. peak and 30 ma. sec.

with W. C. Gamble, and it was agreed that Ward's would fabricate a series of 3 phantoms. In addition, it was decided to make a cooperative study of the practicality of preparing a "chest" phantom of the same basic materials.

As of now, a "hand" (Fig. 3), a "skull" (Fig. 4), and a "dental" roentgen-ray phantom (Fig. 5) have been completed and tested. Since the osseous components are of South Asian origin, the phantoms are approximately 13 per cent smaller and slightly less opaque than their human American counterparts. Nevertheless, comparable roentgenograms were obtained when standard low, medium, and high kilovoltage techniques were employed. In the illustrations shown, the small differences between the roentgenographic densities produced by the phantoms and by the humans at the same exposure level has been increased by photographic reproduction, as is usually the case. Each of the phantoms has proved practical and completely satis-

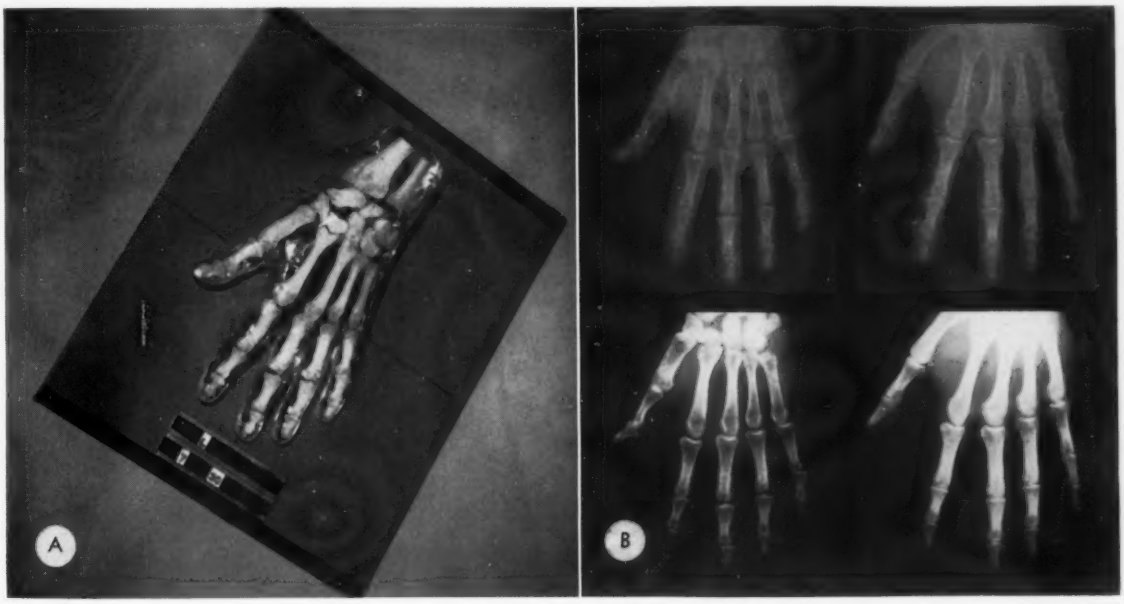

Fig. 3. (*A*) Photograph of "hand" phantom. (*B*) Reproduction of posteroanterior roentgenograms made using the phantom on left and a human hand on right at 40 inches with Hi-Speed intensifying screens. *Upper films:* 90 kv. peak and 0.1 ma. sec. *Lower films:* 46 kv. peak and 2 ma. sec. The phantom and human hand demonstrated almost equal absorption characteristics with both low kilovoltage-high contrast and kigh kilovoltage-low contrast exposures.

FIG. 4. (*A*) Photograph of "head" phantom illustrates use of a head clamp to establish necessary part-film relationship. (*B*) Reproduction of lateral roentgenograms made using human head (16 cm.) on left and phantom (14.5 cm.) on right at 40 inches with a 16:1 grid and Par Speed intensifying screens. *Upper films:* 58 kv. peak and 150 ma. sec. *Lower films:* 80 kv. peak and 35 ma. sec.

factory for the purpose intended.

Good progress is being made on the preliminary steps toward development of the "chest" phantom. It is hoped that a report can be offered within the next year on the success of this phase of the program.

SUMMARY

Durable roentgen-ray phantoms which meet specific requirements have been perfected and made available commercially. They can prove valuable in teaching and studying roentgenographic procedures.

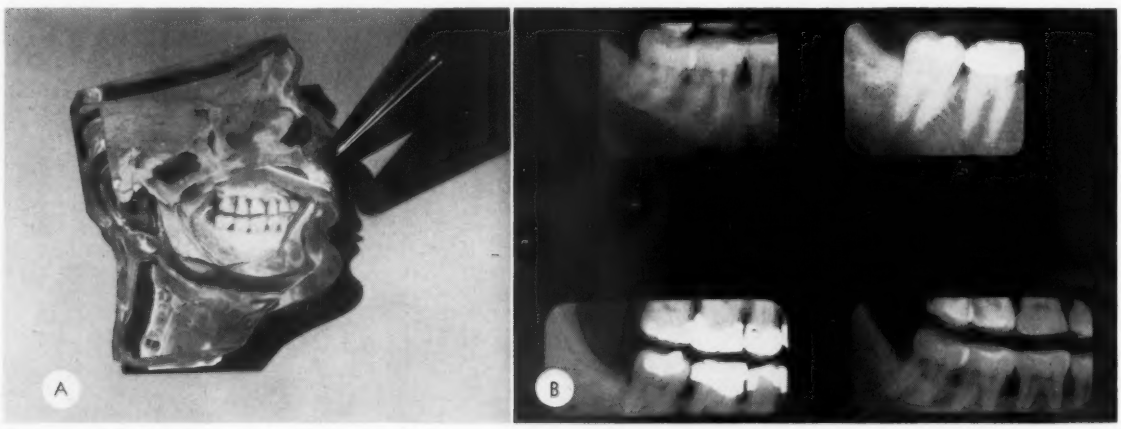

FIG. 5. (*A*) Photograph of "dental" phantom consisting of left lower three-fifths of a skull embedded in Bio-Plastic. Oral cavity is open and teeth separated approximately three-eighths of an inch to accommodate both periapical and bite-wing dental films. Dental film and cone for roentgenographic tube are shown in position to examine upper central incisors. (*B*) Reproduction of periapical and bite-wing roentgenograms using the dental phantom and a human head at 65 kv. peak, 20 ma. sec. and 8 inches distance with Du Pont Type 550 and Type 557 dental roentgenographic films. The roentgenograms which show metallic fillings were made with a living "patient."

Comparative roentgenographic studies and research programs can be facilitated by use of the phantoms or modifications of them. The differences between the absorption coefficients of bone and Bio-Plastic have proved essentially equal to those of bone and human tissue in the 40 to 130 kv. peak range.

Objects can now be obtained which permit both a continuation of research and a reduction in radiation exposure to human beings.

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The author appreciates the encouragement and suggestions given by Dr. J. H. Harris, Jr., and Ward's Natural Science Establishment, Inc.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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Forty-first Annual Meeting: The Homestead, Hot Springs, Virginia, April 6–8, 1959.

EDITORIALS

RADIATION AND THE COMMUNITY: A NEW ROLE FOR THE RADIOLOGIST

THE searing flash and mushroom-shaped cloud at Hiroshima in August, 1945 burned deeply into the consciousness of society a fear of ionizing radiation. Filled with a deep sense of responsibility, nuclear physicists and biologists have been impelled from the quiet of their laboratories into the public forum. Under the dual pressures of anxiety and alarm, these scientists have spoken out strongly concerning the hazards of all types of radiation. In their fields of special competence their opinions must be respected. It is our right, however, to question any public statements on this highly emotionally charged issue which appear to be based upon data having a nebulous quality, upon sheer gross estimates, or upon projection and extrapolation of an apparently clairvoyant nature. We have also the prerogative to speak strongly and affirmatively of our major contributions to humanity as physicians and as pioneers in the recognition of the hazards of radiation.

What position should the radiologist occupy in reference to the radiation hazard problem as it affects his community and the nation? Is his relative muteness to be interpreted as indifference, as timidity, or as ignorance of these issues? How is the medical profession regarded by the general scientific community? These are questions of fundamental importance which we must ask ourselves in this thirteenth year of the atomic age and in the sixty-third year of the use of the roentgen ray in medicine.

Powerful forces of circumstance imperceptibly have cast the radiologist in a new role. He has become a significant link between the physicists and biologists on the one hand, and the general and medical communities on the other. The interpretation, in both a vocal and practical sense, of laboratory findings which have important social consequences are well within his capabilities. The scientific discipline of radiology has given the practitioner the ability to evaluate with sound judgment the potential hazards faced by society from radiation. Unfortunately, this ability is, in general, latent and unexploited.

The community must be given a clear understanding of the position of the radiologist. Publicity concerning radiation hazards has seriously clouded, in the public eye, the legitimate use of radiation for medical purposes. Many persons fail to realize—and this includes a great number of scientists—that we are now in a completely different climate concerning the hazards of radiation than that which existed prior to 1945. Concern with the safety of mankind on a global scale has developed since that year. The control of artificial radiation dosage to within several magnitudes of the natural background level is the projected goal of the health physicists. Had not the march of recent events been so swift, time would have been available to properly digest and assimilate the data relating to the potential hazards of low dosage radiation. The orderly processes of critical examination, confirmation of fact, and rejection of the untrue would have gone on quietly as in the past.

Some thirty years ago no public pressure required the major radiologic organizations of this country to sponsor the National Committee on Radiation Protection.[1] This body, still virtually unknown outside of our specialty, laid the foundations for our

[1] Taylor, L. S. History of the National Committee on Radiation Protection (NCRP), 1956. Unpublished.

Nation's radiation safety measures. General acceptance of its code of suggested standards by radiologists has given their patients and working personnel protection for many years. No legal pressures were necessary. During the past several years both our conscience and intelligence seem to have been consistently underrated.

A nuclear physicist and a chemist[2] in a widely circulated book now carrying the endorsement of a Nobel Prize winning geneticist say: "It is our conclusion, that the excesses in radiation exposure of patients are already too well established in routine practice for them to be corrected intramurally." In a recent issue of the Bulletin of the Atomic Scientists,[3] Professor Z. M. Bacq, Chairman of the United Nations Scientific Committee for the Study of Atomic Radiations, states: "It seems that the medical profession is secretly happy that the main concern of the press and the public is the hazard involved in nuclear weapon tests, and that the important exposure due to radiodiagnostic procedures is rarely considered."

How do we fare in the organized scientific community? We find that in a principal scientific body of this country, representing every major scientific organization, the Committee on Radiation Hazards is headed by a professor of botany. I do not believe that Radiology should have the sole, or even major say in this area, but it is my firm conviction that we possess a breadth of viewpoint which is *not* shared by many who work in more narrow fields. *We must be informed and articulate.*

Where are the roots of the condemnatory viewpoints which have been aired during the past several years? Is any distinction being made between those users of medical radiation specifically trained in this modality and those who are not? The general public—and this includes even men in science—are only recently aware of the radiologist as being the physician who is specifically trained in the use of radiation on human beings. This is a key phrase for the era of radiant energy—*specifically trained in the use of radiation on human beings;* it pinpoints our activities precisely. We are in a unique position, one that offers tremendous possibilities of growth in stature in the public eye, if we proceed wisely and accept the responsibilities which have been thrust upon us.

To what extent are practicing radiologists directly concerning themselves with radiation hazards as they affect the community? Are they spearheading this work at the grass roots level or are they allowing it to go by default to other persons or groups whose background is limited? The results of such a policy, at least in the legislative sphere, can bear bitter fruits. This was evidenced recently in one state by the formation of a radiation control commission politically controlled and motivated. Misguided legislation has also been seen on the Federal level. In the Eighty-fifth Congress a bill was introduced for the compulsory recording of all radiation dosage received by every person in this country.

Our obligations, however, do not end by being concerned solely with those measures having only a direct professional concern. As physicians and as citizens we *must* be informed of the issues involved in the most crucial debate of our time: those dealing with the continued testing of nuclear weapons. The conflict concerning the degree of hazard to human life from radioactive fallout cannot be resolved by us. We can, however, claim some special degree of competence in being able to evaluate those facts which are available. Radiation induced leukemia and neoplasias are, unfortunately, no strangers to our profession. The balancing of human lives against our self interest as a nation involves grave socio-scientific-political decisions. The exercise of judgment in the moral and ethical sphere in a democracy is a perquisite of an informed citizenry. The facts concerning the hazards of radioactive fallout, as well

[2] LAPP, R. E., and SCHUBERT, J. Radiation: What It Is and How It Affects You. Viking Press, 1957.

[3] BACQ, Z. M. The United Nations Radiation Committee. *Bulletin of the Atomic Scientists*, 1958, *14*, 55–57.

as the hazards of radiation used for medical purposes, must be presented in an understandable fashion to the general public. The practitioner specifically trained in the use of radiation is admirably fitted for this purpose, both by virtue of his training and the position of esteem which the physician occupies in his community. In the final analysis, it must be the community and the groups of communities that comprise America which must plan for the nuclear future and which must shoulder any moral burden for the future effects of nuclear testing.

What has been outlined here is not an easy task and there is no clear path to follow. We know, however, that no other scientific discipline has our experience in the use of radiation; no branch of science possesses a comparable experience in dealing with the hazards of radiation; no discipline is so concerned with the interaction of radiation and the whole individual as is ours. If we are fully informed of the complexities of the radiation hazard problem a beginning can be made in the development of proper and far-sighted community programs. The proper beginning of any program is the full utilization by radiologists of existing knowledge to minimize the hazards of radiation in clinical practice. The pattern of protection currently employed by radiologists in a given area can be determined, to some degree, by means of a questionnaire based upon NCRP Handbook No. 60.[4] Such a survey was recently conducted in Washington, D. C., through the Section on Radiology of the Medical Society. This project represented an extremely educational and practical approach for the improvement of protective measures in clinical use. We must be meticulous in our own house before embarking on other programs.

What is to be said about the medical use of radiation outside of the purviews of Radiology? It is not my contention that radiologists alone should handle radiation but that the physician handling this energy must be able to use it safely. This is not a battle of specialists versus nonspecialists, with possible economic overtones, but it is an earnest desire to protect the patient from harm. In this connection, the educational campaign of the American College of Radiology must be mentioned. It is directed to *all* physicians in a sincere desire to protect the American public from radiation hazards by giving information to practitioners who are relatively unaware of the hazards of radiation and the techniques for minimizing them.

To what extent should radiation be used by physicians who do not have an orientation, either scientific or philosophical, in its use? Can radiation be used safely as a fractional part of the activities of a physician? In the face of intensive efforts to reduce radiation exposure to the lowest possible level can we afford—both in terms of individual and of mass population exposure—the indiscriminate use of x-ray equipment? These are questions which the leaders of organized medicine must answer. One of the reasons for the greatness of American medicine has been its ability to police itself. Should not the fluoroscopic or roentgen-ray installations of *all* practitioners be judged by the standards of NCRP Handbook No. 60?[4] Should not *all* practitioners using radiation equipment be completely familiar with the fundamentals of radiation biology and dosage so well expressed in NCRP Handbook No. 59?[5] A roentgen is a roentgen whether it is delivered from the tube of a radiologist or that of another physician. This is an immutable fact and there are no economic overtones to radiation used wastefully in human tissues.

These various facets of the radiation hazard problem thus present a new role for the radiologist. He must be drawn into the public forum, for to stand aside will negate his obligations as a physician and as a citizen. The peaceful use of atomic energy is

[4] X-Ray Protection. National Bureau of Standards Handbook No. 60. Superintendent of Documents, Washington, D. C.

[5] Permissible Dose from External Sources of Ionizing Radiation. National Bureau of Standards Handbook No. 59. Superintendent of Documents, Washington, D. C.

his forte and in these matters he can make a substantial contribution to the community. He can add also to the community understanding of the hazards of radiation used for military purposes. Many years ago, a great American once said: "If there be trouble, let it be in my time, so that my children shall have peace." Perhaps we can not individually or as a group forestall the kind of trouble which Tom Payne could not envisage, trouble which could involve our children to the twentieth generation. However, by the acquisition and dissemination of knowledge in this sphere, the radiologist can exercise a type of leadership consonant with the tremendous contributions to humanity which his specialty has made during this past half century.

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BRIGADIER D. B. McGRIGOR

A HOST of American radiologists deeply feel the loss brought about by the death of Brigadier D. B. McGrigor, *O.B.E.*, M.B., Ch.B., D.M.R.E., Hon. F.I.R.; 55 Park Lane, London W., England.

The Brigadier or "Mac," as he preferred being addressed by his British and American colleagues, is survived by his wife Dorothy, a daughter Heather, and two sons: Dr. A. B. McGrigor, Senior Registrar, Department of Diagnostic Radiology, University College Hospital, London; and Mr. R. B. McGrigor, Senior Registrar, Department of Surgery, St. Bartholomew's Hospital, London, who beginning October, 1958 was attached for one year to the De-

partment of Surgery, University of Michigan Hospital, Ann Arbor.

Born at Torry, Aberdeen, the son of a consulting engineer, McGrigor was educated at Robert Gordon College, Aberdeen, and was graduated from the Aberdeen University in 1907. That year he joined the Royal Army Medical Corps and served in India from 1909 to 1914. He was then transferred to active service in France with the Indian expeditionary force and was placed in charge of a field x-ray section in World War I. In 1918 he was again sent to India as Deputy Director of Medical Services, 9th Indian Division, and specialist in "Electrical Services." In 1920 he was appointed specialist in charge of radiology at Queen Alexander Hospital, Millbank, and lecturer to the Royal Army Medical College, Millbank, London. He retired from active army service in 1927. He immediately became busy on Committees of the British Institute of Radiology; in 1939 he was the Institute's Treasurer and from 1939 to 1942 its President.

In 1939, also, he was called to active army service again as Advisor in Radiology and Chief Consultant to the British Army in Radiology. He organized the Army School of Radiology, located as part of the Royal Army Medical College, Millbank. He was promoted to Brigadier in 1941 and his duties took him on tours of inspection to North Africa, the Middle East and later to the continent. Upon the arrival of the Senior Consultant in Radiology to the American Forces to Great Britain, the tall, genial, Brigadier immediately took him under his wing and was extremely helpful to American Radiologists throughout World War II. It was with the American Senior Consultant that he toured North Africa, Sicily and the Italian front and later the American Army fronts in Europe. These two radiologic consultants narrowly escaped capture when touring the American front together at the beginning of the Battle of the Bulge.

Brigadier McGrigor quickly arranged for American technicians to receive courses in the Army School of Radiology to the end of operating British x-ray equipment. Soon also arrangements were made by the two consultants for American radiologists to have courses in the British Army School of Radiology. Throughout the war the two consultants worked side by side, saving much duplication in equipment and manpower. The British were short of radiologists and the Americans repaid some of the debt to British Army Radiology by loaning American radiologists to the Radiology Section of the Emergency Medical Service of Great Britain which was then

From left to right: Lt. Col. Eric Samuel, the Brigadier's deputy; Dr. Roberts, radiologist from Liverpool, on faculty of British Army Radiological School; Dr. A. E. Barclay, radiologist, in charge of emergency medical service of Great Britain; The Brigadier; Kenneth D. A. Allen, Col. M.C. U. S. Army, senior consultant radiologist E.T.O.; and Dr. R. Boulton Myles, faculty, British Army Radiological School.

conducted by the late A. E. Barclay, who also worked very closely with the British and American consultants in radiology. During this time Brigadier McGrigor's deputy, Lt. Colonel Eric Samuel, spent much time in assisting American radiologists and technicians. Brigadier McGrigor was later decorated with the American Army Legion of Merit and Lt. Colonel Samuel with Bronze Star of the American Army.

Since World War II McGrigor has made two trips to the United States visiting his many American radiologist colleagues and on one of these trips he presented two extremely interesting lectures to the Rocky Mountain Radiological Society meetings in Denver entitled "Forty-Four Years in Radiology in the British Army."

When he retired from the British Army again following World War II, he did not sever his connections with army radiology but remained as secretary and mainspring of the Army X-ray Advisory Committee until it was finally disbanded. When he was made President of the British Institute of Radiology, the membership signified its approval by keeping him in that office for three times the normal term of the office.

He will long be remembered in Great Britain and America as a staunch pillar of radiology, a brilliant, kindly, genial, friendly man who enjoyed nothing better than to sit down to chat with his friends. He was a man whom his friends loved and respected.

Kenneth D. A. Allen, M.D.

NATIONAL COMMITTEE ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

ANNOUNCEMENT OF NEW PUBLICATIONS AND OF WORK IN PROGRESS

Safe Handling of Bodies Containing Radioactive Isotopes (*A Guide for Surgeons, Pathologists, and Funeral Directors*), National Bureau of Standards Handbook 65, issued July 10, 1958.[1] This report is a modified version of Handbook 56, *Safe Handling of Cadavers Containing Radioactive Isotopes*, published in 1953, and includes new information as well as the revised maximum permissible radiation exposure levels developed since then.[2] Pertinent information is given for the guidance of mortuary and medical personnel involved in the handling and autopsy of bodies containing radioactive materials. Individuals performing emergency surgery or autopsy on bodies which had received large internal therapeutic doses of a radioactive isotope are subject to radiation exposure; the handbook discusses the reduction of this occasional exposure to the permissible level. The report indicates that, at least for the next few years, highly radioactive bodies will be encountered only rarely. The body must be adequately tagged so that radiation danger will be recognized by the surgeon or embalmer. Risk depends largely on the relative locations of the isotope and the operative site, and how recently and how large a dose the patient received. Separate sections deal with analysis of extreme cases both with and without opening of the body for surgery or autopsy, accident or injury during surgery or autopsy, contaminated clothing or instruments, cremation, and method of dosage calculation.

Addendum to Report on Protection against Radiations from Radium, Cobalt-60, and Cesium-137 (National Bureau of Standards Handbook 54).[3] A complete revision of Handbook 54 is now in preparation. Until this is completed and in order to bring the present Handbook into conformance with the new maximum permissible radiation levels,[2] a two-page addendum to Handbook 54 has been issued. The changes relate to the new permissible levels, the design of teletherapy apparatus, and radiation hazards resulting from fire.

Consideration of Exposure under Emergency Conditions. Subcommittee 14, "Permissible Exposure Dose under Emergency Conditions" has been working for several years in an endeavor to develop an adequate picture of the effect of large doses of radiation that may be received by large groups of people under emergency or disaster conditions (such as nuclear warfare or the disruption of a large reactor). Doses being considered by this subcommittee are not being regarded as permissible. The report, however, is intended to outline the effects to be expected from large exposures. In the event of nuclear warfare the prime consideration of the population will be immediate survival and therefore large doses may have to be accepted without regard to long-range effects. It is hoped that the Handbook when completed will provide some guidance for the policy decisions that will have to be made under emergency conditions.

The program of this committee was started under the acting chairmanship of L. S. Taylor. The permanent chairman is now Dr. George Leroy, Billings Hospital, University of Chicago, Chicago, Illinois.

REFERENCES

1. Obtainable from Superintendent of Documents, Government Printing Office, Washington 25, D. C., at prepaid cost of 15 cents.
2. Addendum to National Bureau of Standards Handbook 59, obtainable on request from Publications Section, National Bureau of Standards, Washington 25, D. C. or see *Radiology*, 1958, *71*, 263.
3. Holders of Handbook 54 can secure copies of this addendum by writing to Publications Section, National Bureau of Standards, Washington 25, D. C.

SOCIETY PROCEEDINGS AND NEWS ITEMS

NINTH INTERNATIONAL CONGRESS OF RADIOLOGY, MUNICH, JULY 23–30, 1959

The official opening of the Ninth International Congress of Radiology, its Scientific Exhibits and its Commercial Exhibits will take place on July 23, 1959.

The Patron of the Congress is Bundespräsident Prof. Dr. Theodor Heuss; the Honorary President, Prof. Dr. H. Holthusen of Hamburg; the President, Dr. Boris Rajewsky of Frankfurt; and the Secretary-General, Prof. Dr. H. v. Braunbehrens of Munich.

The Scientific Sessions will be held from July 24–30, 1959.

The Preliminary Program has now been issued. The subjects for the General Assemblies include: Image Intensification and Television in Roentgen Diagnosis; Preliminary Irradiation of Malignant Tumors; Radiation Exposure of the Population from Medical Use of Radiation; Automation and Automatics in Diagnosis; Radiation Effect on Cellular Metabolism; Chemical and Biologic Radiation Protection; and Current Radiologic Problems.

The Sectional Meetings will be devoted to diagnostic and therapeutic topics. In addition there will be discussion of questions related to Nuclear Medicine; High Energy Radiation Therapy; Radiobiology; Biophysics; Radiation Hazards and Protection; Physics and Techniques; and The Study and Research of Problems of Radiation Control and Legislation.

In conjunction with this Congress, the new German Roentgen Museum at Remscheid-Lennep will be dedicated.

For further information please address the Secretarial Office of the Congress, 29 Reitmorstrasse, München 22, Germany.

FREEDMAN LECTURES

On Saturday and Sunday, April 11 and 12, 1959, Dr. H. O. Peterson, Professor and Director of the Department of Radiology, University of Minnesota Medical School, will deliver the Eleventh Annual Joseph and Samuel Freedman Lectures in Diagnostic Radiology at the University of Cincinnati College of Medicine. There will be no charge for the lectures.

Radiologists desiring to attend are requested to write Dr. Benjamin Felson, X-ray Department, Cincinnati General Hospital, for further details.

EASTERN CONFERENCE OF RADIOLOGISTS

The Eastern Conference of Radiologists will meet at the Statler Hotel in Washington, D. C., on March 19, 20, and 21, 1959. The host for this meeting will be the Section on Radiology of the Medical Society of the District of Columbia. A balanced scientific program will be presented with equal emphasis on the diagnostic and therapeutic aspects of radiology. A special clinic on therapy problems will be held at the Walter Reed Army Medical Center. As an added note of interest, a trip has also been arranged to the nuclear reactor at the National Naval Medical Center in Bethesda, Maryland.

The social program will begin with a cocktail party on Thursday, March 19. A cocktail party and dinner dance will be held on the following day; in addition, there will be special men's and ladies' luncheons. Special arrangements are also being made for the entertainment of the ladies during the scientific sessions. Further information may be obtained from Dr. U. V. Wilcox II, of 915 19th Street, N.W., Washington 6, D. C.

NEW OFFICERS OF THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

At the Forty-fourth Annual Meeting of the Radiological Society of North America held at the Palmer House, Chicago, November 16–21, 1958, the following officers were elected: President, Laurence L. Robbins, Boston, Mass.; President-Elect, Theodore J. Wachowski, Wheaton, Ill.; First Vice

President, Benjamin Felson, Cincinnati, Ohio; Second Vice President, Alfred M. Popma, Boise, Idaho; New Member of Board of Directors, Robert P. Barden, Philadelphia, Pa.; Chairman, Board of Directors, H. Milton Berg, Bismarck, N. D.; and Secretary-Treasurer, Donald S. Childs, Sr., Syracuse, N. Y.

The Forty-fifth Annual Meeting of the Society will be held November 15–20, 1959, at the Palmer House, Chicago, Ill.

THE AMERICAN CLUB OF THERAPEUTIC RADIOLOGISTS

At the Forty-fourth Annual Meeting of the Radiological Society of North America, held November 16–21, 1958, at the Palmer House, Chicago, Ill., the American Club of Therapeutic Radiologists was founded for the purposes of promoting frequent interchange of ideas among its members and to forward the practice of radiotherapy. There are no plans for special meetings, formal papers, or special publications. Members will meet for a dinner upon the occasion of the meetings of the Radiological Society of North America and the American Radium Society. The following officers were elected: Simeon T. Cantril, Seattle, Wash., President; J. W. J. Carpender, Chicago, Ill., Vice President; and Juan A. del Regato, Colorado Springs, Colo., Secretary.

DIAGNOSTIC ROENTGENOLOGY REFRESHER COURSE

During the week beginning June 15, 1959, the Second Annual Refresher Course in Diagnostic Roentgenology will be held at the Cincinnati General Hospital. The course covers various aspects of diagnostic roentgenology with emphasis on fundamentals. Instruction will be given exclusively by members of the Radiology Staff. Enrollment is limited to radiologists and radiology residents. Tuition is $150.00. Further information and application forms may be obtained from Dr. Benjamin Felson, X-ray Department, Cincinnati General Hospital, Cincinnati 29, Ohio.

POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

A postgraduate course in diagnostic radiology will be presented from March 20–24, 1959 at the University of California Medical Center. The faculty will consist of members of Stanford University and the University of California Departments of Radiology in addition to four distinguished teachers from other parts of the country.

For further details contact: Continuing Medical Education, University of California Medical Center, San Francisco 22, California.

ORINS ANNOUNCES COURSES

The Oak Ridge Institute of Nuclear Studies has announced a series of courses to be held in Oak Ridge, Tennessee, this year. The 1959 curriculum includes courses in the basic techniques of using radioisotopes, courses in the industrial uses of isotopes, and a special summer institute for secondary-school science teachers.

Conducted by the Special Training Division of ORINS, these courses are presented under the organization's contract with the Atomic Energy Commission, and the summer institute is supported by the National Science Foundation in cooperation with the Commission.

The industrial courses, which will be of six weeks' duration, are part of a new program to provide technical personnel from industrial organizations with background for the application of radioisotopes in their own industries. The basic courses last one month, and are devoted to more generalized techniques of radioisotopes and their application in various fields of research and scientific endeavor.

A maximum of 48 participants will be accepted in basic courses and in the summer institute. Industrial courses will be limited to 32.

The one-month summer institute for secondary-school science teachers will be conducted June 29–July 24, with stipends provided for participating teachers. Industrial courses are scheduled February 9–

March 20, and April 6–May 15. Basic courses will open January 5, April 27, May 25, July 27, and August 24.

Additional basic or specialized courses in radioisotopes will be scheduled in 1959 as the need and demand seem to indicate.

Further information and application blanks for any of these courses are available from Dr. Ralph T. Overman, Chairman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee.

THE AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology will conduct an examination on June 2–4, 1959, Palmer House, Chicago, Illinois, The deadline for filing applications is February 15, 1959.

If sufficient interest is shown, a special examination will be offered at this time in Nuclear Medicine to diplomates in Radiology of Therapeutic Radiology. The deadline for filing applications is April 1, 1959.

SYMPOSIUM ON DIAGNOSTIC RADIOLOGY

On March 20 and 21, 1959, there will be a two day symposium on Diagnostic Radiology at the University of California Medical Center, Los Angeles, California, under the chairmanship of Dr. Bernard J. O'Loughlin and co-chairmanship of Dr. Joseph L. Westover. The program will consist of a day and a half of didactic material and a half day session of clinical demonstrations with patients. There will be two guest speakers, Dr. Allan S. Johnston from Leeds, England, and Dr. F. E. Templeton from Seattle, Washington.

Requests for information should be sent to the Department of Continuing Education in Medicine, University of California Medical Center, Los Angeles 24, California.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Röntgenanatomie der Neugeborenen- und Säuglingslunge. By Priv.-Doz. Dr. med. habil. Z. Zsebök, Budapest. Cloth. Price, DM 75.—. Pp. 160, with 286 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

The anatomic basis for the correct interpretation of chest roentgenograms of newborns and infants is the fundamental endeavor of this book of 160 pages. It is emphasized throughout the book that the chest of a newborn is not a miniature edition of a grown-up chest. The very definite differences are stressed.

The different chapters treat the thorax, the lungs, trachea and bronchial tree, the vascular and lymphatic systems, and the mediastinum with special attention to the thymus. Anatomic preparations of fine quality are reproduced and there is a large number of reproductions of chest roentgenograms, most of them normal or deviations from normal and some of them abnormal with reference to anatomic features.

The technical achievement of reproducing photographs and roentgenograms is excellent (there are 286 illustrations).

For radiologists and pediatricians who employ roentgenography of the chest in newborns and infants (and who can read German) the book ought to be a helpful basic guide for the often difficult decision between normal and abnormal.

Hans W. Hefke, M.D.

Le Médiastin et sa Pathologie. By Maurice Bariéty, Professeur de Clinique Médicale à l'Hôtel-Dieu, Membre de l'Académie de Médecine; and Charles Coury, Médecin des Hôpitaux de Paris. Cloth. Price, 11,000 fr. Pp. 854, with 312 illustrations. Masson et Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris 6, France, 1958.

This is the first comprehensive treatise on mediastinal pathology, published in the modern literature (the very first was Hare's monograph, printed in Philadelphia in 1889, which thus appeared prior to the discovery of x-rays by Roentgen). In its *introductory* chapters (pp. 13–52) are recapitulated the essential anatomic, embryologic, and physiologic data. The *second* part includes a discussion of the clinical physiopathology of mediastinal syndromes (pp. 55–85), and of the roentgenologic investigation of the mediastinum (pp. 86–118), with all the "modern" resources, from electrokymography and transverse tomography to roentgen cinematography and pneumomediastinum. The *third* part (pp. 121–382) is devoted to "nonlymphatic, so-called primitive" tumors of the mediastinum, and consists of chapters on "homoplastic dysembryomata" (pp. 152–200), such as bronchogenic cysts and angiomata; on "heteroplastic dysembryomata" (pp. 201–252), for instance teratoids; on neurogenic tumors (pp. 253–278); on thymomata and thymic hypertrophy (pp. 307–350), including as an addendum a special discussion of their association with myasthenia gravis (p. 696); on substernal thyroids and parathyroids (pp. 351–370); on mediastinal ecchinococcus, etc. The *fourth* part (pp. 385–554), entitled "lymphatic diseases of the mediastinum," contains chapters on "sarcoma of the lymphatics and related tumors" (pp. 349–419); on giganto-follicular lymphoma or Brill-Symmers disease (pp. 420–423); on metastatic adenopathies (pp. 424–433); on Hodgkin's disease (pp. 434–457); on sarcoidosis (pp. 457–486); on tuberculous adenopathies (pp. 487–528); and on nontuberculous adenopathies (pp. 529–535). The *fifth* part (pp. 555–600) brings out the diagnostic and other differential points of "false" mediastinal tumors, such as aneurysms of the aorta, pulmonary arteries, or innominate vessels, cardiac enlargement, hiatus hernia, mega-esophagus, esophageal diverticula, and even esophageal varices (when exceptionally large, p. 600), as well as of "true" bronchogenic, esophageal, parietal, and diaphragmatic, tumors. The *sixth* part (pp. 611–695), which treats of mediastinal tissues proper, has sections on acute (pp. 623–637) and chronic (pp. 638–664) mediastinitis; on mediastinal emphysema (pp. 665–679); and on shifts, hernias, and flutter of the mediastinum (pp. 684–695). The bibliography (pp. 699–795) is arranged by subjects, corresponding to

chapter titles. There is also a detailed index, with cross references (pp. 797–843).

This monumental framework is supported by the records of 240 cases of mediastinal pathology, among which 88 were taken from the anatomic collection of Paillas of l'Hôtel-Dieu (on these cases, no roentgenograms, only macro- and microscopic findings were available). Most of the other 152 cases originate from the senior author's personal clinico-anatomic and clinico-roentgenologic files, while some of Coury's cases are identified as such and carry separate numeration. Histologic confirmation could not always be obtained, and in more than a few instances, only a tentative diagnosis was reached. Even so, several chapters are devoid of illustrations, while the reproductions (in positive, *i.e.*, mediastinum in black) are of unequal quality.

Another basic item in the book is the authors' emphasis on diagnostic pneumomediastinum, a procedure which they have employed on over 350 patients, without encountering any serious complications. They consider this method as being part of an "internistic" approach. In fact, although many of their patients underwent exploratory or extirpative operations (most of which were performed by Olivier Monod), the lack of "surgical" experience is evidenced in the merely perfunctory listing of spontaneous rupture of the esophagus (p. 666), or of the problems related to the thoracic ductus (p. 683) (and no mention is made of Killian's thorough analysis of the surgical approaches to the ductus in particular, and to the mediastinum in general, published in 1940 at Leipzig). Conversely, one might object to the liberal space allotted to other conditions, for instance tuberculosis, although this can be easily explained by Bariéty's long standing interest in phthisiology.

Throughout the pages, there is an abundance of semantic offerings, some of which are very pertinent, for instance the differentiation between tuberculoma and granuloma (pp. 522–524). Conversely, these etymologic exercises resulted also in artificious terminology, such as "dysmetabolic mediastinosis" (p. 622), intended to mean deposition of fat tissue (*e.g.*, "local hydrolipopexy") during the time when patient gains weight, which may lead to "chronic adipous mediastinitis." Although they quote Gall and Mallory (second hand, without direct bibliographic indication), they never use the term *lymphomata* (which is the essence of a more unitarian concept), and their classification of these entities is neither modern, nor convenient. Moreover, as far as the follicular varieties are concerned, they do not mention the significant work done in the Army Institute of Pathology and published in 1956 by Rappaport, Winter, and Hicks.

The existence of acute infections in the mediastinum is a generally accepted notion (another omission in their bibliography is the lengthy monograph devoted to this particular subject by Neuhof and Jemerin, published in 1943 at Baltimore). Contrariwise, the so-called "chronic mediastinitis" remains a debatable entity. It is supposed to represent progressive sclerosis in the mediastinum, and may result from almost any process, for instance an inflammation (a mixed "tuberculo-syphilitic" origin is claimed for the case illustrated on p. 653). Less frequent entities are also included, for instance "lipophagic mediastinitis" or paraffinoma, due to spillage of oil from therapeutic intra- or extrapleural applications. The term is used even for malignant situations, for instance the "neoplastic mediastinitis" (p. 660) is said to consist of either or both peritumoral adhesions and cellular infiltrations. Under such circumstances, this reviewer cannot find a justification for the use of the inflammatory suffix *-itis*.

There are many other objectionable items in this treatise on mediastinal pathology, for example the statement that a total dose of 250–500 r *may* be given in treating benign thymic hypertrophy (p. 316). Also otherwise, this work cannot qualify as an educational primer. And yet, its bibliographic background, however incomplete in all but French authors, lists in excess of three thousand titles, all easily accessible. Furthermore, even though it is too seasoned to be palatable for the uninitiated, it can be of considerable value to the discerning reader. With proper discrimination, one may appreciate the many original clinical details included. Mediastinal adenopathy, in the presence of confirmed bronchogenic carcinoma, of confirmed acid-fast infection, or of confirmed infectious mononucleosis, may still be due to associated, not coincidental, nonspecific inflammatory condition; contrarily, when it appears after BCG vaccination, it is much more likely to be of specific nature. For the clinical accuracy of the observations included, and for its wealth of source material, Bariéty and Coury's text is heartily recommended, not as a textbook, but as a reference work for the initiated.

E. R. N. Grigg, M.D.

Roentgenology of the Chest. Edited by Coleman B. Rabin, M.D., F.C.C.P., Attending Physician and Associate Radiologist for Chest Diseases, The Mount Sinai Hospital, New York City; Assistant Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University. Cloth. Price $19.50. Pp. 504, with 247 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1958.

This book, consisting of 464 pages of actual text exclusive of the index, etc., is an admirable diagnostic approach to diseases of the chest in which fifty outstanding clinicians, including at least nineteen radiologists, make notable individual contributions. Although it is obviously impossible to cover all aspects of the complex field of chest diseases in a single volume, the reader will find a surprisingly large measure of valuable information contained herein. Not only are there chapters on important diseases of the lungs, pleura, diaphragm, and thoracic wall, considerable space has also been devoted to disorders of the heart and great vessels.

One inherent fault of edited works which compile essays from numerous invited authors is the lack of a uniform style of writing and a balanced distribution of illustrative material. Dr. Rabin's volume is no exception to this rule, but it has fared better than many other books of the same general type.

Although the book should prove to be extremely useful to any graduate physician desiring a "refresher course" in diseases of the chest, radiologists, chest physicians, and thoracic surgeons in particular will want to add it to their libraries.

John F. Holt, M.D.

Tuberculosis in White and Negro Children. Volume I: The Roentgenologic Aspects of the Harriet Lane Study. By Janet B. Hardy, M.D.C.M. Cloth. Price, $7.50. Pp. 119, with numerous illustrations. Published for the Commonwealth Fund by the Harvard University Press, Cambridge, Massachusetts, 1958.

Tuberculosis in White and Negro Children. Volume II: The Epidemiologic Aspects of the Harriet Lane Study. By Mirian E. Brailey, M.D., Dr.P.H. Cloth. Price, $4.50. Pp. 103, with numerous tables. Published for The Commonwealth Fund by the Harvard University Press, Cambridge, Massachusetts, 1958.

This is a report in two volumes of the Harriet Lane study of tuberculosis in children. This study was based on a sixteen year experience (1928–1944) in a special clinic in Baltimore for young children infected with tuberculosis or in contact with adults having positive sputums, and included 437 white and 892 Negro children.

The first volume concerns the roentgenologic aspects of the study and comprises almost 250 reproductions of roentgenograms with clinical descriptions chosen to illustrate the various phases of childhood tuberculosis. In most cases serial roentgenograms indicate the course of the disease over many years. A few illustrations of other lesions which must be distinguished from tuberculosis are included and the volume also contains a section on bronchography in children.

While the cases presented are very interesting, many of the roentgenograms are of inferior quality, and the emphasis is on the course of the disease rather than on roentgenologic criteria for its diagnosis.

The second volume concerns epidemiologic studies based on the "Harriet Lane" experience —an experience unfortunately confined to the era before specific antimicrobial drug therapy. (New cases were not accepted after 1944 though follow-up studies were continued until 1950.) As one of the authors notes, "It must be remembered that the mortality rates (cited) belong to an era which is already passing in the United States and in other fortunate countries where antimicrobial treatment is available." Certainly the same admonition applies to the roentgenologic aspects of the study.

The work, though meticulously prepared and documented, would appear to be of limited value as a reference volume for the diagnostic roentgenologist. Its interest is already largely historical.

William J. Tuddenham, M.D.

Der Lungenboeck im Röntgenbild. By Prof. Dr. Karl Wurm, Chefarzt privater Kuranstalten in Höchenschwand I. Schw., a. o. Professor für Inn. Med. der Universität Freiburg/Brsg.; Prof. Dr. H. Reindell, Oberarzt der Med. Universitätsklinik, Leiter der Röntgen-Radium-Abteilung Freiburg/Brsg.; and Prof. Dr. Dr. h. c. D. Heilmeyer, Direktor der Med. Universitätsklinik, Freiburg/Brsg. Cloth. Price, DM 78.—. Pp. 220, with 180 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

In the seventy years since Boeck's sarcoid was first described, a huge volume of information has accumulated concerning the dermatologic, ophthalmologic, and general systemic aspects of the disease. For the radiologist, of course, the intrathoracic features of Boeck's sarcoid have been most arresting. Few radiologists, however, have had experience with a series of pulmonary sarcoid cases of any magnitude. Professor Wurm and his associates have carefully studied 300 patients with pulmonary Boeck's sarcoid and present their findings in some detail in this volume. Unlike many collections of this type, however, some order has been made out of the somewhat chaotic complex of roentgen features of pulmonary sarcoidosis. The authors have attempted to differentiate their cases of pulmonary sarcoidosis into stages of the disease which they believe can be followed in logical sequence to various intermediate and end stages. They suggest that by careful analysis of the roentgen features such differentiation is possible, and furthermore that some hint of prognosis may result.

The major portion of the volume is concerned with the presentation of 63 selected cases of pulmonary sarcoidosis in various stages of the disease. Each case is analyzed from the clinical, laboratory, and roentgen aspects in a very complete and instructive manner. Of particular interest are the laminagrams which the authors use liberally to reveal mediastinal and hilar adenopathy and to demonstrate the nature and extent of pulmonary lesions.

Particularly worthwhile and well written is the section on the differential diagnosis of Boeck's sarcoid.

As is usual with German publications the typography and reproductions of roentgenograms are superb.

Certainly anyone interested in pulmonary disease could study this volume with great reward, and having digested its salient features, be prepared to more intelligently analyze future cases of pulmonary Boeck's sarcoid.

Harold Fulton, M.D.

The Atomic Age and Our Biological Future. By H. V. Brondsted. Translated by E. M. Huggard. Cloth. Price $4.75. Pp. 80. Philosophical Library, Inc., New York 16, New York, 1957.

Never before in the history of mankind have we faced possible influences which could cause a general deterioration of the human species. The realization of such an event bears particular importance since we seem to progress further from nature's own law of survival of the fittest as time goes on. Professor Brondsted has written this small concise book for the layman in answer to some of the problems that arise from these considerations. In general, the effects of the atomic age may hold two basic perils for mankind, the first that man may have too little to do and the second that the release of large quantities of radiant energy may have some effect on the genetic constitution of future generations.

These problems are explored primarily from the biologic standpoint in reference to genetic effects. An ethical problem is posed in that future generations may suffer from decisions made today. The possible effect of governmental conscience is only lightly touched upon and perhaps wisely so; the author confines his attention primarily to scientific aspects of the problem. There is an excellently written chapter on the basic characteristics of radiation. Other chapters on elementary biology and genetics help to clarify these subjects. He then proceeds to develop the thesis that mutational effects of genetic alterations resulting from radiation exposure may possibly cause deterioration of the human race. In general, he has created a pessimistic outlook for the atomic age. He may be justified, however, in that dogmatism and dramatism serve to focus a needed attention on problems that might otherwise be minimized. Certainly our genetic heritage must not be taken lightly under any circumstances. Surely every generation ponders technological developments as to their possible deleterious effects on mankind. At each stage of historical development these effects seem to assume more horrendous proportions but none of the earth's peoples before us today have faced the release of forces which could so tragically and completely influence human biologic evolution.

If Brondsted has done nothing more than to create an awareness of these dangers, this book will have adequately fulfilled its purpose.

K. L. Krabbenhoft, M.D.

BOOKS RECEIVED

Lehrbuch der Strahlenheilkunde. Behand'ung mit Röntgenstrahlen und radioaktiven Substanzen. Von Prof. Dr. med. René du Mesnil de Rochemont, Direktor der Strahlenklinik und -Poliklinik der Universität Marburg. Unter

Mitarbeit der Oberärzte der Klinik Dr. H. J. Fiebelkorn, Prof. Dr. E. H. Graul und Doz. Dr. E. Scherer. Cloth. Price, DM 119.—. Pp. 763, with 266 illustrations. Ferdinand Enke Verlag, (14 a), Stuttgart-W, Hasenbergsteige 3, 1958.

The Year Book of Radiology (1958–1959 Year Book Series). Radiologic Diagnosis edited by John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan, and Fred Jenner Hodges, M.D., Professor and Chairman, Department of Radiology, University of Michigan. Radiation Therapy edited by Harold W. Jacox, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia University; Chief, Radiation Therapy Division, Radiologic Service, Presbyterian Hospital, New York City; and Morton M. Kligerman, M.D., Professor of Radiology, Yale University School of Medicine; Director of Radiology, Grace-New Haven Community Hospital. Cloth. Price, $10.00. Pp. 448, with numerous illustrations. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, Illinois, 1958.

Treatment of Cancer and Allied Diseases. Second edition. Vol. I. Principles of Treatment. By fifty-five authors. Edited by George T. Pack, M.D., F.A.C.S., Attending Surgeon, Memorial Center for Cancer and Allied Diseases; Associate Professor of Clinical Surgery, Cornell University Medical College; Surgeon, Pack Medical Group, New York; and Irving M. Ariel, M.D., F.A.C.S., Associate Clinical Professor of Surgery and Associate Attending Surgeon, New York, Medical College, Flower and Fifth Avenue Hospitals; Surgeon, Pack Medical Group, New York. Cloth. Price, $22.50. Pp. 646, with 505 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1958.

Nuclear Explosions and Their Effects. Revised and Enlarged Edition. Cloth. Price, $4.00. Pp. 276. Published by the Director, The Publications Division Ministry of Information and Broadcasting, Government of India and printed at the National Printing Works, 10 Darya Ganj, Delhi, India, 1958.

Tumors of the Lungs and Mediastinum. By B. M. Fried, M.D., F.C.C.P., Associate Attending Physician, Montefiore Hospital; Associate Visiting Physician, Morrisania City Hospital, New York, New York. Cloth. Price, $13.50. Pp. 467, with 231 black and white illustrations, 4 in color on 2 plates. Lea & Febiger, Washington Square, Philadelphia, Pennsylvania, 1958.

Aviation Medicine. Selected Reviews. Edited by Clayton S. White, W. Randolph Lovelace II, and Frederic G. Hirsch, The Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Cloth. Price, $12.00. Pp. 276, with numerous illustrations. Pergamon Press Inc., 122 East 55th Street, New York, New York, 1958.

Annual Review of Nuclear Science. Vol. 8. Emilio Segrè, Editor, University of California, Gerhart Friedlander, Associate Editor, Brookhaven National Laboratory, and Walter E. Meyerhof, Associate Editor, Stanford University. Cloth. Price, $7.00. Pp. 417. Annual Reviews, Inc., Palo Alto, California, 1958.

Unio Internationalis Contra Cancrum Acta. Vol. 13, No. 4–5. Symposium on Cancer of the Liver Among African Negroes in Kampala, Uganda. August, 1956. Cloth. Pp. 873. Published under the Auspices of the International Union against Cancer and with the Financial Assistance of C.I.O.M.S. Prof. J. H. Maisin, Editor-in-Chief, 61, Voer des Capucins, Louvain, Belgique, 1957.

Unio Internationalis Contra Cancrum Acta. Vol. 13, No. 6. Symposium in Leopoldville. September, 1956. Cloth. Pp. 975. Published under the Auspices of the International Union against Cancer and with the Financial Assistance of C.I.O.M.S. Prof. J. H. Maisin, Editor-in-Chief, 61, Voer des Capucins, Louvain, Belgique, 1957.

Nuclear Reactors for Power Generation. Edited by E. Openshaw Taylor, B.Sc., D.I.C., M.I.E.E., M.Am.I.E.E., F.R.S.E. Cloth. Price, $7.50. Pp. 144, with over 50 illustrations. Philosophical Library Inc., 15 East Fortieth Street, New York, New York, 1958.

Elements of Biophysics. By James E. Randall, B.S.E.E., M.S., Ph.D., Associate Professor of Physiology and Biophysics, Department of Physiology and Pharmacology, University of Missouri Medical Center. Cloth. Price, $8.00. Pp. 333. Year Book Publishers, Inc., 200 E. Illinois St., Chicago, Illinois, 1958.

Russian-English Biological & Medical Dictionary. First edition. By Dr. Eugene A. Carpovich. Cloth. Price, $12.00. Pp. 398. Technical Dictionaries Co., Box 144, New York, New York, 1958.

Radiology for Medical Students. Third edition. By Fred Jenner Hodges, M.D., Professor and Chairman, Department of Radiology, University of Michigan; Isadore Lampe, M.D., Professor, Department of Radiology, University of Michigan; and John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan. Cloth. Price, $8.50. Pp. 450, with numerous illustrations. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, Illinois, 1958.

Leitfaden des Strahlenschutzes für Naturwissenschafter, Techniker und Mediziner. Von Dr. med. Hans R. Beck, Facharzt für Radiologie, Lehrbeauftragter für Strahlenschutz am Badischen Staatstechnikum, Karlsruhe; Dr. Rer. Nat. Hans Dresel, Diplomphysiker, Radiologisches Institut der Universität Freiburg/Br.; and Dr. med. Hans-Joachim Melching, Facharzt für Radiologie, Radiologisches Institut der Universität Freiburg/Br. Cloth. Price, $8.60. Pp.

253, with 100 illustrations. Georg Thieme Verlag, (14a), Stuttgart, Herdweg 63, Germany. In the United States and Canada, Intercontinental Medical Book Corporation, New York, New York, 1959.

Acta Radiologica Supplementum 156. Experimental Studies on Radioactive Zinc in the Male Reproductive Organs of the Rat. By Bengt Wetterdal. Paper. Price Sw.Kr. 30:- Pp. 83, with 37 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 157. Radiation Doses to the Gonads of Patients in Swedish Roentgen Diagnostics. Studies on Magnitude and Variation of the Gonad Doses Together with Dose Reducing Measures. By Lars-Eric Larsson. Paper. Price, Sw.Kr. 30:-. Pp. 127, with 32 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 158. Angiocardiographic Observations in Mitral Disease: With Special Reference to Volume Variations in the Left Atrium. By Håkan Arvidsson. Paper. Price, Sw.Kr. 30:-. Pp. 124, with 39 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 159. Percutaneous Selective Angiography of the Coeliac Artery. By Per Ödman. Paper. Price, Sw.Kr. 35:-. Pp. 168, with 49 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 160. Studies on the Effect of Thyrotropic Hormone on the Thyroid Function in Man. By Jerzy Einhorn. Paper. Price, Sw.Kr. 30:-. Pp. 107, with 23 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 161. The Vascular Anatomy of Long Bones. A Radiological and Histological Study. By Gunnar Tilling. Paper. Price, Sw.Kr. 25:-. Pp. 107, with 47 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 162. Renal Angiography during and after Unilateral Ureteric Occlusion. A Long-Term Experimental Study in Dogs. By Torsten Widén. Paper. Price, Sw.Kr. 30:-. Pp. 103, with 37 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 163. Nephrographic Effect and Renal Arteriographic Damage. An Experimental Study. By Carl-Gustaf Helander. Paper. Price, Sw.Kr. 30:-. Pp. 87, with 20 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 164. Congenital Atresia of the Small Intestine. A Roentgenographic Study of 24 Cases. By H. Gladnikoff. Paper. Price, Sw.Kr. 25:-. Pp. 58, with 19 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 165. Coarctation of the Aorta: Aortographic Studies before and after Operation. By Bror Brodén and Johan Karnell. Paper. Price, Sw.Kr. 30:-. Pp. 61, with 37 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Hypophysen-Schilddrüsen-Erkrankungen und endokrine Ophthalmopathie; Radiojoddiagnostik und Strahlentherapie. Von Doz. Dr. med. Wolfgang Horst, Oberarzt am Univ.-Strahleninstitut Hamburg, and Doz. Dr. med. Klaus Ullrich, Oberarzt an der Univ.-Augenklinik Hamburg. Paper. Price, DM 18.—. Pp. 141, with 45 illustrations. Ferdinand Enke Verlag, (14a), Stuttgart-W, Hasenbergsteige 3, 1958.

The Scandinavian Journal of Clinical & Laboratory Investigation. Vol. 10—Supplementum 33. The Synthesis of Prothrombin and Proconvertin in the Reticulo-Endothelial System. An Experimental Study on Rats. By Pär Slätis. Paper. Pp. 83. Helsingfors, Oy Weilin & Göös Ab, 1958.

The Scandinavian Journal of Clinical & Laboratory Investigation. Vol. 10—Supplementum 34. Serological Studies of Human Blood Platelets. By Jon Lundevall. Paper. Pp. 122. Oslo University Press, Oslo, Norway, 1958.

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Porfiria Crónica del Adulto. By Prof. X. Vilanova and Dr. J. Piñol Aguade. Paper. Pp. 70. S. L. Ariel, Barcelona, Spain, 1957.

Sonderausschuss: Radioaktivität. Bundesrepublik Deutschland. Erster Bericht. Paper. Price, DM 4.50. Pp. 68. Georg Thieme Verlag, (14a), Stuttgart, Herdweg 63, Germany. In the United States and Canada, Intercontinental Medical Book Corporation, New York, New York, 1958.

The Love of a Physician, George E. Pfahler, M.D., Pioneer Radiologist. By Muriel B. Pfahler. Cloth. Price, $3.75. Pp. 274 with two illustrations. Dorrance & Company, Inc., Publishers, 131 N. 20th St., Philadelphia 3, Pennsylvania, 1958.

ABSTRACTS OF RADIOLOGICAL LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

López Aydillo, N. R.: A case of Alzheimer's disease. Clinical and histopathologic considerations 356
Naef, R. W.: Clinical features of porencephaly; a review of thirty-two cases 356
Tatelman, M.: The angiographic evaluation of cerebral atherosclerosis 356
Chason, J. L.: Cerebral infarction secondary to occlusive arterial disease 357
Meyer, J. S.: Theory and rationale of anticoagulant therapy in occlusive cerebral vascular disease 357
Webster, J. E., Gurdjian, E. S., Lindner, D. W., and Hardy, W. G.: Neurosurgical aspects of occlusive cerebral vascular disease 358
Tourney, G., Cohen, B. D., and Gottlieb, J. S.: Psychiatric symptoms associated with occlusive cerebral vascular disease 358
Newman, M. K., Jewett, B., and Frampton, G.: Physical medicine and rehabilitation for hemiplegic patients 358
Rowland, L. P., Shapiro, J. H., and Jacobson, H. G.: Neurological syndromes associated with congenital absence of the odontoid process 359

Neck and Chest

Herrnheiser, G.: Roentgen diagnosis of pulmonary edema 359
Tschumy, W., Jr.: Pulmonary infiltration with eosinophilia (Löffler's syndrome) due to smoke inhalation: Report of a case and comment on pathogenesis 359
Walton, E. W.: Giant-cell granuloma of the respiratory tract (Wegener's granulomatosis) 360
Wisoff, C. P.: Bronchiolectasis in chronic bronchitis 360
Harding, H. E., and McLaughlin, A. I. G.: Clinical, radiographic, and pathological studies of lungs of electric-arc and oxyacetylene welders 360
Belcher, J. R.: Large air cysts of the lung ... 361
Schlager, K.: Postpneumonic pseudo-air-cysts in children 361
Sniffen, R. C., Soutter, L., and Robbins, L. L.: Muco-epidermoid tumors of the bronchus arising from surface epithelium 362
Scheer, R.: Cinedensigraphy, a photoelectric method in the differential diagnosis of pulmonary carcinoma 362
Hallerbach, H., and Schaede, A.: The diagnosis of complete transposition of the pulmonary veins 362
Torner-Soler, M., Balaguer-Vintró, I., and Carrasco-Azemar, J.: Cardiac dextroposition: Hypoplasia of the right pulmonary artery with right venous pulmonary drainage into the inferior vena cava 363
Jürgens, R., Stecken, A., and Witte, H.: The clinical significance of roentgenologic demonstration of wall calcifications in the region of the left atrium 363
Steinberg, I.: Pericarditis with effusion: New observations, with a note on Ewart's sign.. 363
Monk, I., Reeve, T. S., Kalokerinos, J., and Wingfield, J.: A preliminary report on a study of experimentally produced tricuspid and mitral valve regurgitation 364
Klein, M. M., and Walsh, E. L.: Relation between cardiac failure and gastric ulcer in the older age group 364
Schoch, G.: The silhouette sign 364
Schäfer, H., and Westerkamp, H.: Cinematographic observations on diaphragmatic tic of childhood 364
Svoboda, M.: The oesophagus in pneumomediastinography 365
Kaufmann, H. J.: X-ray findings in a child with Candida esophagitis 365
Wagner, J. M., and Kessel, I.: Complications of *Candida albicans* infection in infancy. 365
Bernstein, L. M., Pacini, R., Fruin, R. C., and Gorvett, E.: Esophagitis as a cause of upper abdominal pain 366
Lorenz, D.: The significance of dysphagia in surgical conditions of the oesophagus; with some comments on operative treatment.... 366
Da Rocha, R. M., and Tavares de Macedo, L. E.: Esophageal and gastric stenosis due to ingestion of acid in a case of attempted homicide 367

ABDOMEN

SHEHADI, W. H.: Radiological and geographical considerations in the diagnosis of peptic ulcer 367

CANDARDJIS, G.: On the value of certain indirect signs in the radiologic diagnosis of the stomach 367

PINKERTON, P.: Psychogenic megacolon in children: The implications of bowel negativism 368

MARINE, R. and LATTOMUS, W. W.: Cavernous hemangioma of the gastrointestinal tract; report of a case and review 368

VARADARAJAN, M. G.: Interposition of a loop of ileum between the dome of diaphragm and liver 368

BURNETT, W., and SHIELDS, R.: Movements of the common bileduct in man; studies with the image intensifier 369

SALZMAN, E., WATKINS, D. H., and RUNDLES, W. R.: Opacification of radiolucent biliary calculi 369

VERSTRAETEN, J.: Postcholecystectomy 369

WOLF, H. G.: Contributions to the diagnosis and etiology of spontaneous pneumoperitoneum in the newborn 370

GYNECOLOGY AND OBSTETRICS

STEIN, I. F., SR.: The Stein-Leventhal syndrome; a curable form of sterility 370

HUTCHINSON, D. L., BENNETT, L. R., and GEAN, D. A.: Isotopic localization of the placenta in placenta previa 370

GENITOURINARY SYSTEM

MAYER, R. F., and MATHES, G. L.: Retrocaval ureter 371

COPPRIDGE, A. J., and RATLIFF, R. K.: Unilateral multicystic kidney disease 371

LEWIS, S., and DOSS, R. D.: Calcified hydropyonephrosis 371

HENRIQUES, C. Q.: Osteomyelitis as a complication in urology; with special reference to the paravertebral venous plexus 371

PERILLIE, P. E., and CONN, H. O.: Acute renal failure after intravenous pyelography in plasma cell myeloma 372

PALUBINSKAS, A. J., and HODSON, C. J.: Transintervertebral retroperitoneal gas insufflation 372

SWART, B., and SCHÄFFER, A.: Symptomatology and diagnosis of primary retroperitoneal tumors 372

NERVOUS SYSTEM

REINHARDT, K.: The advantages of combining abrodil myelography with diskography ... 373

SKELETAL SYSTEM

WILLICH, E.: The roentgen sign of the translucent metaphyseal lines in the newborn .. 374

ROENTGEN DIAGNOSIS

HEAD

LÓPEZ AYDILLO, N. R. Acerca de un caso de enfermedad de Alzheimer. Particularidades clinicas e histopatológicas. (A case of Alzheimer's disease. Clinical and histopathologic considerations.) *Bol. Inst. Pat. Med.*, July, 1958, *13*, 181–187. (From: Sección de Histopatología del Sistema Nervioso del Instituto Cajal y del Servicio de Neuropsiquiatría del Hospital Provincial, Madrid, Spain.)

Alzheimer's disease is the most representative of the presenile diseases. This name should be reserved for cases genetically conditioned by individual constitution and considered as an exaggeration of the physiologic processes of senility.

Numerous reports of cases related with systemic degenerative diseases of adolescence (familial spastic paralysis, Hallervorden-Spatz disease, etc.) and with postencephalic syndromes (including posttraumatic cases) have been published under the name of Alzheimer's disease.

At the present time, this last number of conditions is grouped together under the name of "Acquired Symptomatic Complex of Alzheimer." The differentiation is sometimes impossible due to the similarity of the histopathologic picture in all these cases having in common cerebral atrophy.

The clinical diagnosis is very difficult to arrive at, due to the lack of specific symptoms which may entirely simulate those of an organic form of dementia.

However, there are numerous histopathologic changes which have been reported after Alzheimer's original description, which today are considered as characteristic of Alzheimer's disease. These are: net and argentophile corpuscle of Csermerly; granulovacuolar degeneration of Sinchowicz and those described by Rio Ortega in the subependymal region. Therefore, the only certain method of diagnosis of Alzheimer's disease during life would be cerebral biopsy. The cerebral atrophy which can be demonstrated by pneumoencephalography is nonspecific and worthless in the differential diagnosis. The author makes no mention of his experience with cerebral biopsy.—*Jorge Thompson, M.D.*

NAEF, RICHARD W. Clinical features of porencephaly; a review of thirty-two cases. *A.M.A. Arch. Neurol. & Psychiat.*, Aug., 1958, *80*, 133–147. (From: The Department of Neurology, Jefferson Medical College, Philadelphia, Pa.)

A series of 32 cases of porencephaly is presented with emphasis on the clinical features of the disease. The term porencephaly is used to indicate cystic defects in the cerebrum communicating with the ventricles and/or the subarachnoid space. The author limits the term porencephaly to those cases which are due to a developmental malformation or to a destructive lesion which occurred prior to, incidental to, or shortly after birth.

The clinical features of proencephaly usually consist of symptoms referable to a focal cerebral hemispheric lesion. The most common symptoms are spastic weakness, seizures, delayed speech development, homonymous visual defects, delayed unilateral limb growth and mental retardation. The onset of symptoms was within the first year of life in 14 of the 32 patients, but the early clinical picture was so mild that in the majority of cases it did not precipitate adequate study until adolescence or later. Symptoms appear in a cumulative manner and the clinical course is progressive in most of the patients.

Roentgenograms of the skull were abnormal in 8 of the 32 patients. In 6 individuals there was rarefaction or thinning of the parietal bone which corresponded to the porencephalic lesion. In one patient there was enlargement of the calvarium on the side of the cyst while in another individual there was hypoplasia of the calvarium on the involved side.

Pneumoencephalography revealed the porencephalic defect in all but one patient. In this case a large cyst was found at exploration, but it did not appear to communicate with either the ventricle or the subarachnoid space. All other cysts communicated with the lateral ventricle and in 3 instances there also appeared to be a communication with air in the enlarged subarachnoid spaces adjacent to the cyst.

The location of the cyst corresponded to the lobe indicated clinically in 22 of the 32 patients. In the others the cyst location did not appear to correlate with the clinical signs and symptoms, suggesting that there is considerable brain damage in addition to the porencephaly.

Electroencephalography was performed on 26 patients and was abnormal in 22. In 14 cases the electroencephalogram disclosed foci corresponding closely to the location of the cyst.

Treatment was not very effective although in some instances there was improvement in seizures following anticonvulsive therapy or excision of the cysts.—*J. L. Williams, M.D.*

TATELMAN, MAURICE. The angiographic evaluation of cerebral atherosclerosis. *Radiology*, June, 1958, *70*, 801–810. (Address: 1420 St. Antoine St., Detroit 26, Mich.)

The purpose of this paper is to call attention to the roentgenologic manifestations of cerebral atherosclerosis which was found to be the cause of stroke symptoms in 58 per cent in a series of 200 consecutive cases with a clinical diagnosis of "stroke" or "cerebral vascular accident." In all cases angiograms were obtained by percutaneous injection of 8 to 10 cc. of 50 per cent hypaque with from 2 to as many as 8

injections being made, in most cases under local anesthesia only.

Complete occlusion of the internal carotid artery was found in 10.5 per cent of this series with most of the occlusions occurring immediately above its origin in the neck. Occlusion of the middle cerebral artery was found in only 4.5 per cent, with the obstruction close to its origin in most of the cases. Surprisingly, 7.5 per cent of the series showed nonfilling of one anterior cerebral artery. While it has been thought that such findings represent some variation in vascular dynamics or a congenital narrowing or absence of the anterior cerebral artery, the author feels that many, if not all, of these cases are actually caused by atherosclerosis, with either complete occlusion of the vessel, or narrowing of its lumen to the point where blood flow is insufficient to allow it to be filled by contrast injection. Occlusion of the vertebral-basilar system was found in 5 cases, 2.5 per cent of the total series.

Partial occlusion of the lumen of the cerebral vessels is manifested by an eccentric, often broad based, filling defect in the lumen caused by the plaque. Minor changes in the vessels may show only a slight irregularity in the vessel lumen with or without narrowing, dilatation, abnormal tortuosity, straightening, and elongation of the vessels. These changes in the internal carotid artery are usually quite localized, while the changes in the intracerebral branches tend to be more generalized or involve longer segments of the vessels.

The author emphasizes the relative safety of the angiographic procedure and its importance in cases of "stroke" as the location of an incomplete occlusion may permit surgical removal of the atheromatous plaque which in time will often cause a more serious complete occlusion.—*Donald N. Dysart, M.D.*

Chason, J. L. Cerebral infarction secondary to occlusive arterial disease. *Radiology*, June, 1958, *70*, 811–814. (Address: 1401 Rivard St., Detroit 7, Mich.)

Noninfectious cerebral infarcts are of 2 kinds: the anemic type in which the secondary hemorrhage is limited to the periphery of the infarcted area; and the hemorrhagic type where blood fills most or all of the area destroyed. The occlusive arterial disease responsible for the infarct may be either atherosclerosis, thrombosis, or embolism.

Anemic infarcts generally produce a typical sequence of events. As softening and liquefaction gradually develop, the ensuing fluid attraction and cell infiltration produce enlargement of the infarcted area. This is most evident about the second or third week and may result in clinical signs of an expanding mass lesion. Roentgenograms at this time may disclose a shift of the pineal gland and pneumoencephalograms may reveal a decrease in the size of the adjoining arachnoid and ventricular spaces and even displacement of the ventricular system. Generally, these changes begin to regress by the end of the third week, resulting in a decrease in size of the infarcted area and focal enlargement of the adjacent ventricular system. If the adjacent ependyma is destroyed an acquired porencephalic cyst will develop. The hemorrhagic infarct exhibits a similar sequence, but produces earlier and greater enlargement and persists for a longer time.

Several intravascular complications may occur distal to an occluding lesion. These include: capillary engorgement and anoxia with subsequent necrosis or endothelial hypertrophy and hyperplasia; the development of arterial or capillary emboli within the infarct; and the proximal and distal extension of an occluding thrombus or embolus or the superimposition of a thrombus upon an obstructing atherosclerotic plaque.—*Edward B. Best, M.D.*

Meyer, John S. Theory and rationale of anticoagulant therapy in occlusive cerebral vascular disease. *Radiology*, June, 1958, *70*, 815–824. (From: Department of Neurology, College of Medicine, Wayne State University, Detroit, Mich.)

It is generally conceded that long-term anticoagulant therapy with heparin and dicumerol is of benefit in thrombosis and occlusion of the internal carotid and basilar arteries. Anticoagulant therapy reduces the incidence of the attacks, but does not appear to affect the course of severe infarction once it has occurred. Origina ly it was thought to be due to recanalization of the occluded vessel. An alternate possibility is that the anticoagulant drugs result in an improvement in the collateral circulation supplied by small arterial anastomoses after occlusion of a major vessel.

The author reports the results of an investigation of the action of anticoagulant drugs in experimental cerebral vascular disease. The middle cerebral artery was occluded with asceptic technique in 23 monkeys and 11 cats. In half these animals no anticoagulant drugs were used and in the other half coagulation was suppressed with either heparin or dicumerol. The clotting time was maintained between forty and ninety minutes and the prothrombin time between forty and eighty seconds. Severe ischemia or infarction was produced by lowering the blood pressure at the time the cerebral artery was occluded. A sterile modified Forbes window in the bone flap permitted study of the ischemic area with a high-power microscope. In some animals saturated solution of trypan blue was injected intravenously to demonstrate any change in vascular permeability. In others, filtered India ink was injected into the femoral vein to indicate any tendency toward agglutination of small particles. Localized plasma concentration changes were studied by intravenous methylene blue. Meas-

urements of oxygen tension were made by the polarographic method.

The mechanism of localized changes in cerebral vascular resistance following vascular occlusion is first slowing of local blood flow with ischemic anoxia. This results in endothelial damage and localized loss of fluid constituents of the blood, with subsequent brain edema and localized hemoconcentration, followed by increased adhesiveness of all formed elements of the blood, of the endothelium, and of the fine carbon particles introduced into the circulation. The administration of heparin and dicumerol prevents the adhesiveness of red cells, white cells, and platelets. By this means it prevents increased vascular resistance and promotes better collateral circulation.—*Arno W. Sommer, M.D.*

WEBSTER, J. E., GURDJIAN, E. S., LINDNER, D. W., and HARDY, W. G. Neurosurgical aspects of occlusive cerebral vascular disease. *Radiology*, June, 1958, *70*, 825–830. (Address: J. E. Webster, 801 David Whitney Bldg., Detroit 26, Mich.)

The surgeon's interest in cerebral vascular disease began with his concern for hemorrhagic collections on the surface of the brain—the epidural and subdural hematomas. With the development and use of cerebral angiography since the thirties, other cerebral vascular lesions have gained increasing attention. More recently, patients having a so-called "cerebral vascular accident," or "stroke syndrome," have been referred to the neurosurgeon for evaluation, with collaboration by the radiologist in the performance of cerebral angiographic studies, in seeking a physiologic explanation for the dysfunction associated with cerebral vascular insufficiency and/or failure. In an analysis of the first 200 patients so studied, a multiplicity of lesions described as "strokes" have been found. Slightly more than 10 per cent of the patients had surgical mass lesions including brain tumor, intracerebral hematoma, subdural hematoma, and brain abscess. Occlusive carotid artery disease as a cause for cerebral vascular insufficiency and failure was found in about 20 per cent of the cases. Occlusion of other arterial trunks and branches accounted for another 35 per cent, and the remainder included such conditions as embolism, cerebral hemorrhage (hemorrhagic infarction), saccular aneurysm, cerebral atrophy, encephalitis, etc.

A valuable and safe test for cerebral vascular insufficiency in the presence of hemiparesis or hemiplegia has been the use of carotid artery compression. Upon such compression, syncope may occur, indicating a contralateral complete or partial carotid occlusion, an anterior cerebral artery occlusion, or basilar artery involvement. A sensitive carotid sinus reflex may mask these responses. Syncope does not usually occur in patients with mass lesions or occlusion of the middle cerebral artery trunk or its branches.

The treatment is directed primarily toward the approximately 10 per cent of these patients who have a mass intracranial lesion rather than cerebral vascular disease. Patients with complete carotid artery occlusion have been treated surgically by excision of a segment of the involved artery and a cervical sympathectomy. Any measure which increases collateral circulation through the external carotid system would seem to be worthwhile. Patients with segmental atheromatous involvement of the bulb and the internal and external carotid at their origins have been treated by an intimectomy, with removal of the plaques and stenotic areas of the lumina following previous treatment by carotid compression to induce collateral flow and by anticoagulant therapy. Anticoagulant therapy is also used in the treatment of anterior cerebral, middle, and basilar occlusions before cerebral vascular failure with paralytic signs occurs. The patients are also taught to use carotid compression daily to dilate and increase vascular channels toward improving collateral blood flow. Both physical and psychological rehabilitation are also highly important.—*W. H. Jarvis, Jr., M.D.*

TOURNEY, GARFIELD, COHEN, BERTRAM D., and GOTTLIEB, JACQUES S. Psychiatric symptoms associated with occlusive cerebral vascular disease. *Radiology*, June, 1958, *70*, 831–834. (Address: G. Tourney, 951 E. Lafayette, Detroit 7, Mich.)

Twenty-four patients suffering from occlusive arteriosclerotic cerebral vascular disease were studied. This number included 7 patients with right anterior cerebral artery occlusion, 8 with left anterior cerebral artery occlusion, 1 with left middle cerebral artery occlusion and 2 each with right internal carotid, left internal carotid, bilateral carotid and possible vertebral artery occlusion. The area of involvement was determined by careful neurologic study and cerebral angiography. Psychiatric symptoms and the results of various psychologic tests were evaluated and analyzed. These were correlated with the angiographic-neurologic findings. The results are presented in tabular form.

The following conclusions are drawn: (1) there is no specific psychiatric syndrome related to involvement of any one area; (2) there is a suggestive positive relationship between age and the degree of intellectual deficit; and (3) there is a consistent exaggeration of premorbid personality traits following the occlusion. A patient with premorbid depressive traits becomes more consistently depressed following the occlusion. The same applies to anxiety symptoms, hypochondriasis, impulsivity and delusions.—*Z. Petrany, M.D.*

NEWMAN, MAX K., JEWETT, BARBARA, and FRAMPTON, GERALD. Physical medicine and rehabilitation for hemiplegic patients. *Radiology*, June, 1958, *70*, 835–840. (Address:

M. K. Newman, 16801 Wyoming, Detroit 21, Mich.)

Recent advances in medical and surgical techniques have markedly improved the hitherto bleak outlook of brain-damaged patients. This has become a matter of great importance in view of our steadily increasing geriatric population. In terms of disability, the cerebrovascular accident far outranks coronary sclerosis although the latter is a more prevalent cause of death. The authors outline an integrated plan of treatment for restoring the hemiplegic patient physically, mentally and socially. First, an evaluation is made of the patient's neurologic, psychiatric, medical, and social status. As an example, prior to re-education, an examination is made to elicit the following information: (a) manual muscle test of strength, coordination, and spasticity at all joints; (b) extent of flaccidity; (c) severity of dysarthria and aphasia; (d) impairment of special senses; (e) state of motivation; (f) associated diseases; and (g) local joint phenomena.

In re-educating paralyzed muscles, the more primitive or gross motions are restored before the finer movements, and therapy is geared along this line. Training procedures in ambulation include: (1) balance training; (2) alleviation of joint pain by means of physiotherapy; (3) release of spasticity; (4) muscle re-education; (5) occupational therapy; (6) gait training; (7) general conditioning to attain stamina and endurance; and (8) speech therapy, if indicated. It is estimated that with modern methods of physical medicine and rehabilitation, over 90 per cent of brain-damaged patients can be made self-sufficient, learning to ambulate and care for their personal needs. This frees them from prolonged residence in the hospital or nursing home and restores the patient to the family group.—*John S. Alexander, M.D.*

Rowland, L. P., Shapiro, J. H., and Jacobson, H. G. Neurological syndromes associated with congenital absence of the odontoid process. *A.M.A. Arch. Neurol. & Psychiat.*, Sept., 1958, *80*, 286–291. (Address: H. G. Jacobson, Montefiore Hospital, 210th St. & Bainbridge Ave., New York, N. Y.)

Two cases are presented showing an association between absence of the odontoid process of the second cervical vertebra and cervical myelopathy. A total of 17 cases is analysed. Atlanto-axial dislocation was demonstrated in at least 11 of the cases studied and the authors believe that excessive mobility at this joint may cause trauma to the spinal cord or to its blood vessels and meninges.

Clinical manifestations are divided into four groups:

Group 1 includes 7 cases with severe cervical pain following trauma and an absence of subsequent neurologic signs.

Group 2 includes 5 cases of trauma in which transient weakness occurred. In only one of these cases was there any permanent neurologic abnormality.

Group 3 cases presented slowly progressive signs and symptoms of cervical myelopathy with no history of trauma.

Group 4 cases had solely cerebral symptoms.

It is suggested that congenital absence of the odontoid process is more than a curiosity and that its recognition deserves more emphasis.—*Richard E. Kinzer, M.D.*

Neck and Chest

Herrnheiser, G. (Deceased). Zur Röntgendiagnostik des Lungenödems. (Roentgen diagnosis of pulmonary edema.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Aug., 1958, *89*, 125–135.

More than twenty-five years ago, Felix developed a new concept of the lung structure. He separated the pulmonary lobe into lobe root, center and cortex. The findings were confirmed by previous studies of the author.

In this paper, Herrnheiser discusses the anatomic details and the roentgenologic appearances. Many pathologic processes affect exclusively or at least to the greater part only the cortex, others are limited to the center. There are probably different functions of cortex and center. It is perhaps important to note that the inner central lobules are supplied by narrow caliber branches of the pulmonary arteries which originate directly from the comparatively large secondary or tertiary lobar arteries. The lobar center and parahilar region are not identical. A narrow cortical zone extends between the lobar centers to the lung roots. Consequently cortical processes may occur at the hilus.

Densities predominantly or entirely localized in the lobar centers produce the butterfly shadows which have long been known as typical for some types of pulmonary edema. The peculiar shape of the densities can be explained by the concept that the pathologic processes involve the lobar centers.

On analyzing normal and pathologic pulmonary markings, one should distinguish cortical and central phenomena. The composition of the lung root by two elements, namely the lobar roots and hilar structures, provides the basis for a logical subdivision of the hilar shadows.

Reproductions of 14 roentgenograms and 8 anatomic illustrations and drawings accompany the text for easier understanding of the new theoretic concept.—*Hans Lewin, M.D.*

Tschumy, William, Jr. Pulmonary infiltration with eosinophilia (Löffler's syndrome) due to smoke inhalation: Report of a case and com-

ment on pathogenesis. *Ann. Int. Med.*, Sept., 1958, *49*, 665–672. (From: The Department of Internal Medicine, University of Texas Southwestern Medical School, Dallas, Texas.)

A case of transitory pulmonary infiltration and eosinophilia (Löffler's syndrome) following smoke inhalation is reported. It is postulated that this patient had an allergic response in the lungs to an unknown antigen in the smoke. Complete and dramatic resolution followed the administration of prednisone.—*James J. McCort, M.D.*

Walton, E. W. Giant-cell granuloma of the respiratory tract (Wegener's granulomatosis). *Brit. M. J.*, Aug. 2, 1958, *2*, 265–270. (From: Department of Pathology, Queen's College, Dundee, Scotland.)

This is a report of 10 patients with a widespread fatal granulomatous disease, which has recently come to be known as Wegener's granulomatosis. Forty-six additional cases are summarized from the literature.

The disease occurs in young or middle-aged adults of either sex. The onset is insidious with symptoms of persistent nasal or pulmonary infection. Malaise, fever and weakness are marked. The course is usually rapid, progressing to death in as little as four weeks or as long as five years, with an average of five months. There may be temporary improvement following antibiotic treatment but the inflammatory reaction continues and causes extensive mucosal ulceration, widespread pulmonary consolidation, and destruction of the bone and cartilage of the nose and soft palate. In late stages, evidence of widespread inflammatory disease occurs and there is often high fever, arthralgia, a hemorrhagic rash, and a terminal uremia or bronchopneumonia.

The pathologic process consists of extensive ulceration throughout the respiratory tract with necrosis of cartilage and bone. The histologic examination shows granulomatous inflammation with giant cells and necrosis. The lesions are widespread and are most often perivascular. There are also extensive vascular lesions with focal necrosis in the small arteries and arterioles of many organs, especially the kidney, lung, and spleen. The lesions in the vessels resemble polyarteritis nodosa, and the author believes that many cases of Wegener's syndrome have been reported as polyarteritis nodosa.

The roentgenologic changes in the lungs are of particular interest. In slightly over one-half of the cases dense circular or oval opacities are present in one or more lobes. These vary in size up to 4 cm. in diameter and there is often central cavitation. Less often, bronchopneumonic infiltration is the only significant roentgen change. In all but 2 of the 56 reported cases the chest roentgenograms were abnormal. Unfortunately, only one roentgenogram is shown and the roentgen aspects are discussed very briefly.

The etiology of the initial respiratory tract disease is unknown. The author believes that the widespread secondary lesions are due to some type of hypersensitivity mechanism.

Treatment is primarily directed toward healing of the initial lesion, and roentgen therapy in low dosage has proved successful in some cases. Administration of cortisone or other steroids is the treatment of choice once widespread lesions have developed.—*J. L. Williams, M.D.*

Wisoff, Carl P. Bronchiolectasis in chronic bronchitis. *Radiology*, June, 1958, *70*, 848–850. (Address: Norfolk General Hospital, Norfolk 7, Va.)

Bronchiolectasis is a manifestation of chronic bronchitis characterized by saccular dilatation of the terminal bronchioles, often associated with emphysema. Zorini and Pigorini first recorded the condition as "larval bronchiectasis" in 1934. They reported the clinical picture as one of chronic bronchitis in which hemoptysis is a prominent symptom. The chest roentgenograms were negative, but the bronchograms showed small club-shaped distal bronchi. Simon and Galbraith, in 1953, described the bronchogenic findings in 90 patients with a clinical diagnosis of chronic bronchitis. The bronchographic observations consisted in dilatation, diverticulosis, and narrowing of the bronchi and pooling of the contrast medium in the terminal bronchioles.

The author presents 2 cases of bronchiolectasis which were demonstrated by bronchography. In one case bronchograms showed peripheral pooling of contrast material in the terminal bronchioles, involving almost all segments. This patient's dyspnea was apparently due to the widespread involvement and nonfunction of the respiratory surface. The second case showed pooling only in the right upper lobe. This man gave a history of asthma-like wheezing and chronic cough for about thirteen years.—*Arno W. Sommer, M.D.*

Harding, H. E., and McLaughlin, A. I. G. Clinical, radiographic, and pathological studies of the lungs of electric-arc and oxy-acetylene welders. *Lancet*, Aug. 23, 1958, *2*, 294–398. (Address: Director of Pathology, Salisbury Area, Salisbury, England.)

Details are given of the lungs of four electric-arc welders and one oxygen-acetylene cutter who came to necropsy. The authors state that as far as they could find out these men have never been exposed to silica dust. Their findings indicate that considerable quantities of pure iron oxide dust can be inhaled and remain deposited in the lung tissue without producing fibrosis. The slight degree of fibrosis which may

be present in the lungs of some electric-arc welders is probably due to some other constituents of the welding fume. Sufficient fibrosis is not present even in welders with long experience to cause any disability, unless they have been exposed to extraneous sources of silica.

The retained dust gives roentgenographic shadows that may be indistinguishable from fibrotic pneumoconiosis.—*Arthur E. Childe, M.D.*

Belcher, J. R. Large air cysts of the lung. *Brit. J. Clin. Pract.*, Feb., 1958, *12*, 87–93. (From: London Chest Hospital, London, England.)

The author discusses large air cysts of the lungs and describes 3 types, each of which may become so distended with air as to cause mechanical interference with respiration.

Infantile Tension Cysts. Infantile lobar emphysema and infantile tension cysts are probably manifestations of the same pathologic process. In one, the presence of a large cyst dominates the macroscopic appearances, but emphysema is found in the rest of the affected lobe. In the other, the emphysema predominates, but small cysts may be present. The condition appears to be due to some form of check valve mechanism, permitting the lobe concerned to be blown up and leads to the breakdown of alveolar walls. Exact cause of the check valve mechanism is not known.

Symptoms depend upon the size of the developing cysts and vary from occasional episodes of dyspnea to severe cyanosis and respiratory distress. Gross mediastinal displacement away from the affected side is the dominant feature as demonstrated by the roentgenogram.

Anepithelial Cysts. These cysts are probably due to a congenital abnormality which causes a check-valve mechanism. It is suggested, however, that in the anepithelial cysts the mechanism operates farther out in the bronchial tree and so leads to involvement of a much smaller part of the lung than seen in the infantile tension type.

The cysts are attached by a narrow stalk to the hilus of the lung. There is apparent communication with the bronchial tree, but it is sometimes impossible to demonstrate. They are unlike emphysematous bullae in that they can be removed in one piece and lie under the visceral pleura, whereas the visceral pleura itself forms the outer wall of emphysematous cysts. The inner wall is devoid of epithelium.

Symptoms seldom develop before adult life. The lesions therefore have to become very large before the mechanical interference with respiration, which they cause, leads to dyspnea. Infection is rare and gradually increasing dyspnea is a prominent symptom. Pneumothorax may develop.

Emphysematous Cysts. The etiology of emphysematous cysts is not fully known, but they are a part of an essentially degenerative and generalized disease. The fundamental lesion is a breakdown of alveolar walls, leading to communication of one with another and to breakdown of interstitial planes. The process is gradually progressive with destruction of wide areas of the lung. The visceral pleura, being a strong membrane, does not give way and so comes to form the outer wall of the cysts. The blood vessels and bronchi are resistant to the destructive process and they persist to form trabeculae.

Emphysematous cysts seldom cause symptoms before the fifth decade. The patients give a history of long standing bronchitis with a wheeze and a periodically productive cough. Progressive dyspnea is the predominant symptom, sometimes aggravated by the development of a spontaneous pneumothorax.

The presence of a large air containing cyst is not on its own an indication for surgery. The disease is not always progressive, nor do the cysts necessarily increase in size. Symptoms of increasing respiratory distress and repeated spontaneous pneumothoraces justify operation.—*James F. Martin, M.D.*

Schlager, K. Über postpneumonische Pseudoluftzysten beim Kind. (Postpneumonic pseudo-aircysts in children.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Aug., 1958, *89*, 136–146. (Address: St. Petergasse 13, Wien 17, Austria.)

Postpneumonic pseudo-aircysts (PPC) represent air containing cavities in the pulmonary parenchyma which develop in the course of inflammatory, mostly pneumonic, processes. The terminology in the publications since 1927 varies. The author believes that the term "postpneumonic pseudo-aircysts" is the best, since it differentiates the PPC from the congenital lung cysts and gives an important hint of the etiology. In addition, it gives the impression of a thin-walled cyst-like air-containing cavity. Most authors believe that a valve-like stenosis of a small bronchus or bronchiolus causes these cavities in the lung periphery. It is difficult to prove organic bronchostenosis, although it has been reported in the literature. Since organic changes have rarely been found, the occurrence of functional bronchostenosis must be suspected.

A valve-like bronchostenosis causes dilatation of the alveoli and lobuli by increase of pressure. This is a functional emphysema with the possibility of complete regression. On the other hand, there may be the possibility of cavities developing by poststenotic overinsufflation of purulent gangrenous tissue.

The author reports the following findings as characteristic for PPC: (1) roentgen-proven or at least anamnestic pneumonia; (2) round or oval ring-shadow; (3) thin wall, as if drawn by a pencil; (4) if the PPC is larger than the original infiltration, it cannot be explained by simple abscess clearing; this

also applies if the PPC is smaller than the original infiltration, occurring in a lung after resolution without residual infiltration; (5) significant changes of size, especially enlargement in asymptomatic patients; (6) displacement, mostly not very marked; and (7) complete regression without any special therapy.

The differential diagnosis is difficult since paucity of clinical symptoms is the rule.

The etiology and diagnosis of the postpneumonic pseudo-aircysts are discussed on the basis of the author's 4 cases, with references to the literature.—*Hans Lewin, M.D.*

SNIFFEN, RONALD C., SOUTTER, LAMAR, and ROBBINS, LAURENCE L. Muco-epidermoid tumors of the bronchus arising from surface epithelium. *Am. J. Path.*, July–Aug., 1958, *34*, 671–684. (From: The Department of Pathology, Surgical Services, and Department of Radiology of the Massachusetts General Hospital, Boston, Mass.)

Five cases of intrabronchial neoplasm apparently arising from surface respiratory epithelium are described. In each case the symptoms were those of slowly developing bronchial obstruction, with symptomatic history up to thirty years' duration. Serial roentgenograms showed signs of bronchial obstruction or increase in size of a mass. The mass was usually slightly lobulated, homogeneous in density, and resembled a peripheral primary carcinoma or isolated metastatic neoplasm. The physical examination was not helpful, except when a complicating pneumonitis was present.

Both gross and microscopic structure of the 5 tumors resected suggested relatively benign neoplasms. They were intrabronchial and did not penetrate beyond the cartilaginous rings. They grew along the bronchus into its branches and expanded it. They bore no microscopic resemblance to either carcinoid or cylindromatous types of bronchial adenoma, or to the adenomas arising in mucous glands.—*A. H. Hall, M.D.*

SCHEER, R. Die Cinédensigraphie, eine photoelektrische Methode zur Differentialdiagnose des Lungenkarzinoms. (Cinedensigraphy, a photoelectric method in the differential diagnosis of pulmonary carcinoma.) *Die Medizinische*, March, 1958, No. 11, 422–429. (From: Chirurgische Klinik der Universität des Saarlandes, Homburg, Saarland, Germany.)

Cinedensigraphy is a method in which 2 photoelectric cells register the pulsations of the capillaries of the lungs. Each cell explores one lung and the resulting curve is compared with the curve of the contralateral lung. Simultaneous electrocardiograms are taken since the findings vary with the systolic and diastolic phases of the heart. Because the amplitude of the curve is dependent on the thickness of the thorax, prior standardization is absolutely essential.

A curve is pathologic: (1) if the amplitude is smaller than normal or is entirely absent; (2) if pronounced changes occur, such as a slow instead of the normal quick rise; or if the peak is rounded, flat, or kinked instead of pointed; and (3) if the pulsations are so-called "mixed," that is, when the pulsations of the ventricles, great vessels, and capillaries are intermingled.

Cinedensigraphy can distinguish primary from metastatic lesions in the lungs, for primary lesions show no pulsations at or adjacent to the site of the lesions, whereas metastatic lesions produce pulsations even when the lesion is fairly large. Squamous cell carcinomas and oat cell tumors produce curves with a markedly decreased amplitude. Peripheral bronchogenic carcinomas and reticulum cell sarcomas produce no pulsations. Lymphosarcomas show no diminution in pulsation. All tuberculous lesions fail to cause an increase or decrease in pulsations. Pleurisy, dry or wet, causes a localized decrease. Pulmonary fibrosis and emphysema show a bilateral decrease in amplitude. An old lung abscess causes a diminished amplitude but a fresh abscess will have a normal curve.

In 200 cases previously proved as malignant by bronchoscopic study, cinedensigraphy was positive in 95 per cent. In another 200 cases, in which bronchoscopy, roentgenography, bronchography, angiopneumography, and bronchial aspirations showed negative findings, cinedensigraphy indicated neoplasm in 90 per cent and was subsequently confirmed by surgery and histologic section. False positives may occur, but their number can be reduced by meticulous attention to standardization. It should also be noted that tachycardia produces inaccurate results, therefore cardiac rate should be decreased by medication prior to the investigation by cinedensigraphy.—*J. Zausner, M.D.*

HALLERBACH, H., and SCHAEDE, A. Die Diagnostik der kompletten Lungenvenentransposition. (The diagnosis of complete transposition of the pulomary veins.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Aug., 1958, *89*, 152–171. (Address: H. Hallerbach, Strahlenklinik Prof. Dr. R. Janker, Baumschul-Allee 12–14, Bonn, Germany.)

The diagnosis of pulmonary vein transposition, *i.e.*, the drainage of the pulmonary veins into the right heart, has been increasingly important since this anomaly has to be differentiated from the atrial septum defect which is amenable to surgery. With the introduction of the extracorporeal circulation, surgical reimplantation of the transposed pulmonary

veins comes into consideration. Further diagnostic problems are: differentiation between partial and complete pulmonary vein transposition, topography of the pulmonary vein anomaly, and recognition of concomitant defects and complications.

The authors give a very comprehensive description of the embryologic, anatomic, and pathophysiologic fundamentals of the anomaly, with reference to the publications on the subject in the literature.

Several schematic drawings illustrate the distribution of arterial and venous blood under normal conditions, in partial and incomplete transposition of the pulmonary veins. Another drawing shows the oxygen content of the blood in different parts of the heart and in the great vessels, as found by cardiac catheterization and arterial puncture. Still another schematic drawing shows the oxygen saturation in the atria and in the great veins, in different types of complete pulmonary vein transposition.

After this introduction, the authors discuss the diagnosis of *complete* transposition of the pulmonary veins in the light of their experience with 20 of their own cases. The description is accompanied by numerous excellent reproductions of roentgenograms of the chest, a few kymograms, roentgenograms of the heart and great vessels with indwelling catheters, laminagrams (body section roentgenograms), and angiocardiograms.

For diagnosis, an identical O_2-percentage in the right atrium and in the peripheral arteries is a *conditio sine qua non.*

For localization of the anomaly, roentgen examination—including angiocardiography and cardiac catheterization—is necessary. Even with all these elaborate methods, localization can be difficult. Only in cases in which the pulmonary veins drain into a persistent left superior vena cava and the left vena anonyma in the upper vena cava, are the findings always characteristic.—*Hans Lewin, M.D.*

Torner-Soler, M., Balaguer-Vintró, I., and Carrasco-Azemar, J. Cardiac dextroposition: Hypoplasia of the right pulmonary artery with right venous pulmonary drainage into the inferior vena cava. *Am. Heart J.*, Sept., 1958, *56*, 425–430.(From: The Cardio-Angiology School of the University of Barcelona, Barcelona, Spain.)

The case reported in this article is that of a twenty-two year old woman who had no symptoms referable to the circulatory system. She was thought to have a dextrocardia on routine physical examination. There was some bulging of the left hemithorax. She had a Grade 3 systolic murmur in the whole right precordial region and a soft diastolic murmur which sounded like a collateral circulation murmur.

Chest roentgenograms showed overdistention of the left lung and displacement of the heart to the right. The left pulmonary artery was large and pulsated vigorously. The right pulmonary artery was small and showed no pulsation. An abnormal shadow in the right lung ran parallel to the right inferior heart border and seemed to become broader as it approached the diaphragm. It had the appearance of a vascular shadow and as it broadened near the base of the lung it seemed more likely that it was venous. The electrocardiogram ruled out dextrocardia. There was right ventricular hypertropy.

The angiocardiogram showed a large left pulmonary artery which carried most of the blood, and hypoplasia of the right pulmonary artery. The shadow along the right inferior heart border was a large pulmonary vein emptying into the inferior vena cava. The possibility of an associated atrial septal defect was not excluded.

The classical signs are shown in this case. They are pulmonary systolic murmur with a continuous (collateral) diastolic murmur, along with a large distended left lung and pulmonary artery and a small right lung and pulmonary artery. The anomalous venous drainage into the inferior vena cava should be suspected when a vascular shadow which widens inferiorly is seen just to the right of the heart.—*Frank M. Windrow, M.D.*

Jürgens, R., Stecken, A., and Witte, H. Die klinische Bedeutung des röntgenologischen Nachweises von Wandverkalkungen im Bereich des linken Vorhofs. (The clinical significance of roentgenologic demonstration of wall calcifications in the region of the left atrium.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1958, *88*, 534–545. (From: Röntgenabteilung der I. Medizinischen Universitätsklinik und Poliklinik der Charité, Berlin, Germany.)

The findings in the authors' own 4 cases and 38 cases from the literature are in complete agreement.

Calcification in the left atrium is usually associated with mitral stenosis and considerable degree of insufficiency, auricular fibrillation, and multivalvular disease of long duration. This finding is a contraindication to surgery for mitral stenosis.

Laminagraphy in 2 planes is the method of choice for demonstration of left atrial calcifications.—*F. M. Reis, M.D.*

Steinberg, Israel. Pericarditis with effusion: New observations, with a note on Ewart's sign. *Ann. Int. Med.*, Aug., 1958, *49*, 428–437. (From: Department of Radiology, The New York Hospital-Cornell Medical Center, New York, N. Y.)

In the past it was thought that pericardial effusions accumulate primarily in the infracardiac and posterior portions of the pericardial sac and that filling of the lateral portions occurs only in massive effusions. This was considered logical because it was assumed that the heart was fixed in its position by

the great vessels above and the inferior vena cava below. Corresponding with this idea, Ewart described an area of dullness at the inferior angle of the left scapula in pericardial effusions. This was accompanied by bronchial breathing, increased fremitus and egophony in the same area. He attributed these findings to compression of the lung tissue and bronchi by the distention of the pericardial sac with fluid. Pericardial taps through the left posterior chest wall became popular.

Angiocardiograms show that a major part of a massive pericardial effusion does accumulate in the anterior or retrosternal portion of the pericardial sac and that it actually does push the heart posteriorly. Because much of the fluid is anterior in position and one does not have to traverse lung tissue, the anterior approach below the xyphoid process is recommended for pericardial tap.—*Frank M. Windrow, M.D.*

MONK, IAN, REEVE, THOMAS S., KALOKERINOS, JAMES, and WINGFIELD, JOHN. A preliminary report on a study of experimentally produced tricuspid and mitral valve regurgitation. *Brit. J. Surg.*, July, 1958, *46*, 75–82. (From: The Unit of Clinical Investigation, The Royal North Shore Hospital of Sydney, Sydney, Australia.)

This study is an attempt to apply a new concept to the problem of restoring anatomic continuity to a fibrosed and distorted mitral or tricuspid valve.

Ewes were used as experimental animals. Valvular regurgitation was produced. The postoperative presence of regurgitation was confirmed by cardiac catheterization and selective angiocardiography.

Baffles were created in the atria proximal to the deformed valves. These were produced by infolding of the atrial walls. Pressure recordings and angiocardiograms are shown to indicate that the baffles create a zone of resistance to lessen the high velocity backflow of mitral and tricuspid regurgitation.—*James J. McCort, M.D.*

KLEIN, MICHAEL M., and WALSH, EUGENE L. Relation between cardiac failure and gastric ulcer in the older age group. *Radiology*, May, 1958, *70*, 672–683. (Address: M. M. Klein, VA Hospital, Huntington, W. Va.)

Of 1,187 cardiac patients seen in a six-year period, excluding rheumatic and valvular heart disease, 370 (31.2 per cent) had gastrointestinal symptoms of sufficient degree to warrant roentgen examination. In 115 of this number, the findings were positive for gastric ulcer. The ages of the patients ranged from twenty-seven to ninety-two, the average being sixty-three years.

Of 193 patients with hypertensive heart disease without failure, there were 34 (17.1 per cent) with gastrointestinal symptoms, and positive ulcer findings in 7 (3.6 per cent of the total group). Arteriosclerotic heart disease and coronary heart disease are closely related and showed an almost identical result. Of 333 patients with arteriosclerotic heart disease without failure, 99 (29.7 per cent) complained of gastrointestinal disturbances, and there were positive ulcer findings in 28 (8.6 per cent of the total group). Of the 343 patients with coronary heart disease without failure, 128 (37.3 per cent) had gastrointestinal complaints and in 40 (11.7 per cent of the total group) the roentgen findings were positive for gastric ulcer. There were also 318 cases examined with cardiac failure without regard to the basic type of heart disease, with 94 (29.6 per cent) having significant gastrointestinal complaints, and positive ulcer findings in 40 cases (12.4 per cent of the total group). During a subsequent period 53 consecutive cardiac failure cases received roentgen examination on admission or as soon afterward as possible, regardless of the type of heart disease, and regardless of whether or not gastrointestinal symptoms were present. Fifteen (28.3 per cent) of the 53 had gastric ulcers, which is a much higher incidence than in the earlier group of failure cases. The course of the gastrointestinal symptoms and the gastric ulcer paralleled the cardiac status.

There is no more controversial subject in the medical literature than the pathogenesis of gastric and duodenal ulcer. To the more familiar etiologic factors there probably should be added cardiac decompensation of various degrees and arteriosclerotic vascular disease.

Brief abstracts of 10 cases are presented.—*Walter H. Jarvis, Jr., M.D.*

SCHOCH, G. Das Silhouettezeichen. (The silhouette sign.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1958, *88*, 503–520. (From: Röntgendiagnostisches Zentralinstitut des Kantonsspitals, Zürich, Switzerland.)

The silhouette sign, first described by B. Felson and H. Felson, is discussed and illustrated by schematic drawings and roentgenograms. If a soft tissue tumor and a thoracic organ are in anatomic contact and at nearly the same distance from the film, their respective contours will be lost in the area of contact on the roentgenogram. The silhouette of the organ is changed by the shadow of the tumor. If, however, the lesion and organ are at different levels from the film, then the contour of the organ remains distinct. Thus, it is possible to determine the location of a lesion in the sagittal plane and in relation to the central thoracic organs from a posteroanterior roentgenogram.—*F. M. Reis, M.D.*

SCHÄFER, H., and WESTERKAMP, H. Kinematographische Beobachtungen beim Zwerchfell-

Tic im Kindesalter. (Cinematographic observations on diaphragmatic tic of childhood.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1958, *88*, 521–525. (From: Kinderklinik der Freien Universität Berlin, Kaiserin-Auguste-Victoria-Haus, und Röntgenstitut Dr. H. Westerkamp, Berlin-Neukölln, Germany.)

Cinematographic observations in 3 cases of diaphragmatic tic are described. In unilateral involvement, normal diaphragmatic movements alternate with jerky, paradoxical movements of the hemidiaphragm. In bilateral involvement, alternate rapid upward movements of the hemidiaphragm are seen, resembling the two sides of a scale. The alternation between normal and "tic" movements differentiates this from other abnormal movements of the diaphragm.

Diagnosis is possible from knowledge of the symptoms and by fluoroscopy.—*F. M. Reis, M.D.*

Svoboda, Milan. The oesophagus in pneumomediastinography. *Radiol. clin.*, May, 1958, *27*, 161–169. (From: Department of Radiology, Institute of Haematology and Blood Transfusion, Prague, Czechoslovakia.)

The author's combined method of pneumomediastinography and contrast filling of the esophagus enables him to demonstrate the inner and outer contours and the mucosal pattern of the esophagus. Fifty cases have been examined by the combined method.

The indications for the combined examination are all esophageal tumors and esophageal diseases of questionable diagnosis and in which surgery is considered. The contraindications are severe disorders of the cardiovascular and respiratory systems, esophageal perforation and mediastinitis.

The author's technique consists of preliminary clinical examination of the cardiovascular system and routine roentgenograms of the thorax. The patient is fasted for twelve hours and no premedication is employed. After routine skin preparation and subcutaneous anesthesia of the suprasternal notch, an intravenous needle is inserted in the midline, from 0.5 to 1.0 cm. above the upper border of the manubrium, 0.5 cm. deep. The tip of the needle is pointed caudally to the dorsal side of the manubrium and inserted from 1.0 to 2.0 cm. deep. By aspiration with an attached syringe it can be determined whether a vessel has been injured.

In the examination of the cervical esophagus, from 300 to 500 ml. of nitrogen monoxide is employed. In the case of the thoracic esophagus, from 600 to 1,200 ml. of the gas is injected. The insufflation is carried out by special equipment under fluoroscopic control and by adjusting the position of the patient and the tilting of the table. After the pneumomediastinum is produced, barium suspension or paste is given to the patient by mouth. The patient is kept in bed for twelve hours after the examination.

In the author's series, there were no complications. The patient experiences a feeling of retrosternal pressure and a change in voice because of the emphysema of the neck. The nitrogen monoxide is completely absorbed in from twenty-four to forty-eight hours. Five cases are illustrated by reproduction of roentgenograms.—*J. N. Ané, M.D.*

Kaufmann, Herbert J. X-ray findings in a child with Candida esophagitis. *New England J. Med.*, June 5, 1958, *258*, 1143–1146. (From: The Departments of Pediatrics and Radiology, Boston City Hospital, and the Department of Pediatrics, Boston University School of Medicine.)

The author describes the roentgen appearance of esophageal candidiasis in a four year old boy who died after prolonged therapy with cortisone and antibiotics for a refractory aplastic anemia. Autopsy revealed *Candida albicans* infection of the esophagus and systemic spread to the lungs and spleen.

The roentgenographic findings are summarized. Early cases show involvement of the lower portion of the esophagus, with a normal appearance of the upper ½ to ¾. In severe cases of long standing the entire esophageal mucosa may be involved. The changes affect the outline of the esophageal mucosa with the production of irregularly shaped protrusions and indentations of varying sizes. The longitudinal folds may be preserved but, with severe involvement, are no longer detectable. They disappear in the lower segment first. In severe involvement, narrowing of the esophageal lumen may be noted, again beginning in the inferior portion of the esophagus.

The mural changes caused by *Candida albicans* in the esophagus closely resemble those due to esophageal varices. The differential diagnosis must include esophageal varices, bacterial esophagitis and chemical or thermal esophagitis. *Candida albicans* infection is largely limited to debilitated patients and those receiving antibiotics or steroids or both.—*James F. Martin, M.D.*

Wagner, J. M., and Kessel, I. Complications of *Candida albicans* infection in infancy. *Brit. M. J.*, Aug. 9, 1958, *2*, 362–366. (From: Department of Paediatrics, University of the Witwatersrand, Transvaal, U. of S. Africa.)

While thrush is a common infection among sickly and undersized infants, the infection can spread to: (1) the gastrointestinal tract, especially the esophagus; (2) the lungs; and (3) the blood stream, with widespread lesions.

Esophagitis is a serious complication. Pathologically, the process is the same as in the mouth with

patches of greyish-white membrane on the mucous surface, the patches consisting of a tangled growth of the organisms. Destruction of epithelium follows and there is often secondary bacterial growth. Involvement of the esophagus leads to dysphagia and resultant inhalation bronchopneumonia. Clinically, the infant is toxic, dehydrated and cyanotic on eating. Thrush esophagitis is a reasonably common disease. It can and should be diagnosed and treated during life.

Four cases are reported in patients from fifteen days to nine months of age. Lipiodol swallow examination showed a spill-over into the trachea, irregularity in outline of the esophagus, and cardiospasm. Chest roentgenograms revealed extensive bronchopneumonia. Two patients recovered with resolution of the roentgenographic findings. Two patients died and necropsy on one showed the typical thick white granular membrane covering the mucosal surface of the esophagus. *Candida albicans* was recovered from the membrane.—*Leslie K. Sycamore, M.D.*

Bernstein, Lionel M., Pacini, Ralph, Fruin, Robert C., and Gorvett, Edmund. Esophagitis as a cause of upper abdominal pain. *J.A.M.A.*, Sept. 6, 1958, *168*, 27–33. (From: The Gastroenterology Section, Medical Service, and Research Division, Veterans Administration Hospital, Hines, Ill.)

A controlled acid perfusion test has been used on 20 patients with a diagnosis of abdominal pain of undetermined cause. It was also used on an additional 13 patients with abnormal gastrointestinal roentgen findings. The test indicated an esophageal origin of symptoms limited to the abdomen in patients with inflammatory changes who had no positive roentgen findings. A positive response in a patient with symptoms but without confirmatory endoscopic findings is considered a case of pseudoesophagitis. Possibility of microscopic abnormalities is presumed in these cases.

The authors believe that interpretation of the negative responses is most important. Positive tests are always helpful. In 4 of the 13 patients with roentgen evidence of gastrointestinal disease, abdominal symptoms originated in the "X-ray normal esophagus" rather than in the organ known to be diseased. The test is clinically valuable, but should be simplified to make it more practical.—*R. E. Kinzer, M.D.*

Lorenz, D. The significance of dysphagia in surgical conditions of the oesophagus; with some comments on operative treatment. *Germ. med. Monthly*, Aug., 1958, *3*, 254–258. Original article in the *Deutsche med. Wchnschr.*, 1958, *83*, 1081. (From: The Department of Surgery, University of Freiburg, Freiburg, Germany.)

Dysphagia may be a manifestation of functional as well as organic disease. Carcinoma makes up two thirds of all the disorders of the esophagus. In the author's series of 207 patients with dysphagia, there were 167 cases of carcinoma. Painless dysphagia is the first, the most characteristic and often the sole symptom in carcinoma of the esophagus. As long as only a portion of the esophageal wall is infiltrated, obstruction to the passage of food is not prominent. Symptoms of obstruction rapidly become more severe once the whole circumference of the esophagus is involved.

Resection and esophagogastrostomy produce the best results if the lower third of the esophagus is involved by carcinoma. In the middle and upper thirds, the results of surgery are not so good because of the early involvement of the regional lymph nodes and anatomic difficulties. Rotational radiation therapy has a definite place in these cases. In inoperable stenosing carcinoma of the esophagus or cardia, early gastrostomy is indicated.

Benign tumors of the esophagus are rare. A gradual onset of dysphagia which may persist for many years is characteristic. Eventually, marked dilatation with painless dysphagia, retrosternal pressure, dry retching and vomiting results.

The differential diagnosis of benign and malignant tumors may be difficult. Esophagoscopy is of considerable value in this respect. The benign pedunculated tumors may be removed endoscopically and the benign tumors of the wall are enucleated extramucosally with good results.

Traction diverticula of the esophagus are frequently discovered as symptomless findings in routine roentgenologic examination. Vague dysphagia at the beginning of meals and retrosternal discomfort after rapid drinking are the main features. Retention of food may result in esophagitis. Small traction diverticula do not necessitate surgery. The large diverticula require operation for relief of obstruction.

Pulsion diverticula during the early course produce irritating cough and expectoration of tenacious sputum. Fluids cause more trouble than solids. Regurgitation of undigested food occurs during and immediately after meals. Complete esophageal obstruction may result when the diverticulum becomes filled. Surgery is the procedure of choice.

Scar formation following extensive inflammatory mediastinitis results in a fixed narrowed esophagus and dysphagia. Surgery is of considerable value in the relief of these cases.

Primary mediastinal tumors seldom produce dysphagia, because they displace the esophagus only. In the presence of a right-sided aorta the esophagus may be compressed between the vessels with resulting dysphagia. Enlargement of the mediastinal lymph nodes may also produce dysphagia. In tuberculous mediastinal lymphadenitis the differential diagnosis from nonspecific lymph node enlargement depends

upon the history. Metastatic mediastinal lymph node enlargement, especially from bronchial carcinoma, produces dysphagia more commonly than does inflamatory lymphadenitis.

Foreign bodies impacted in the esophagus produce immediate dysphagia.

Cardiospasm occurs more commonly in women between the ages of thirty and fifty years. The onset of symptoms is gradual. Roentgenologically, there is secondary dilatation of the esophagus above the constriction which tapers to the cardia.

Esophagitis results from reflux flow of the acid gastric juice due to lack of closure of the cardia sphincter. It also follows esophagogastric anastomoses. Esophagitis frequently complicates hiatal hernia and congenital shortening of the esophagus.

Peptic ulceration of the esophagus occurs more frequently in the lower third in islands of heterotopic gastric mucosa. Ulcerations associated with esophagitis are usually superficial, whereas true peptic ulcers tend to be deep. Roentgenologically, the mucosa around the ulcer may appear edematous. The mucosal folds may converge at the site of the ulcer.—*J. N. Ané, M.D.*

Da Rocha, Roberto Martinho, and Tavares de Macedo, Luiz Emílio. (Rio de Janeiro, Brazil.) Estenose esofagiana e gástrica por ingestão de acido; (a propósito de um caso de tentativa de homicídio). (Esophageal and gastric stenosis due to ingestion of acid in a case of attempted homicide.) *Rev. brasil. med.*, May, 1958, *15*, 319–322.

Esophageal injury by corrosives is either inadvertent (mostly in children) or suicidal (the largest number in this category are females in the fifteen to twenty-five year age group, of low social and cultural standing, but with great emotional problems).

An exception to the above rule is the case of a twenty-three year old white Portuguese female who was brought to the emergency room of a hospital in Rio de Janeiro. She alleged that, as she was strolling alone at night, four unknown men stopped, assaulted, and raped her, and afterwards forced her to drink a caustic liquid, presumably with the intent to kill her. NaOH is usually neutralized by the gastric juice, but this patient must have ingested a solution containing a strong acid (perhaps sulphuric or hydrochloric acid) because both the esophagus and the stomach suffered severe burn, with subsequent stenotic sequels.

Partial gastrectomy and repeated esophageal dilatations were successful in restoring functionally the patient's upper alimentary tract. Periodic follow-up roentgenologic examinations are contemplated for the remote possibility of late malignant transformation of esophageal or gastric scar tissues.—*E. R. N. Grigg, M.D.*

Abdomen

Shehadi, William H. Radiological and geographical considerations in the diagnosis of peptic ulcer. *Schweiz. Ztschr. f. allg. Path. & Bakt.*, No. 2, 1958, *21*, 345–354. (From: Department of Radiology, New York Polyclinic Medical School and Hospital, New York, N. Y.)

The author states that a crater shadow is the only conclusive diagnostic evidence of a peptic ulcer. Indirect evidence such as swelling and irregularity of the mucosal folds, irritability and deformity may be observed in conjunction with an ulcer crater, but the findings are not diagnostic because they occur in many conditions other than peptic ulcer. Excessive spices and condiments, alcoholic beverages, and strong coffee can cause transient irritability and swelling of the mucosa. Parasitic infestation, especially ankylostomiasis, may cause ulcer-like symptoms. Roentgenologically, there is swelling and irregularity of the duodenal mucosa, irritability, and spasticity with increased peristaltic activity. The changes are reversible following a vermifuge.

The author emphasizes that peptic ulcer is basically a personality problem, generally among urban dwellers. Ankylostomiasis is essentially an economic and public health problem, occurring most frequently in rural dwellers with low standards of living. However, other individuals may be accidentally exposed during travel or in the unsanitary conditions of war. Consequently, personality types, eating habits, geographical distribution and social standards must be taken into consideration when there is indirect evidence suggestive of a peptic ulcer but no crater is found by the radiologist.—*J. L. Williams, M.D.*

Candardjis, G. Sur la valeur de quelques signes indirects en radio-diagnostic gastrique. (On the value of certain indirect signs in the radiologic diagnosis of the stomach.) *Radiol. clin.*, May, 1958, *27*, 148–160. (From: Institut universitaire de radiologie de Lausanne, Lausanne, Switzerland.)

The importance and definitiveness of "direct" signs of organic lesions of the stomach are too well known and accepted to call for any further evaluation or reiteration.

However, the value of "indirect" signs, though not specific, should not be underestimated. Usually they are considered an accompaniment of benign lesions. At most they suggest only a probable diagnosis.

While functional deformities (spasm, hourglass deformity, etc.) may be transitory, they serve as a "pointing finger" to an organic lesion which may not yet be recognizable or is otherwise overlooked.

The author takes exception to the teaching that

ulcerative lesions of the greater curvature are malignant and maintains that benign greater curvature lesions are more frequent than is ordinarily accepted.

The author cites 4 cases in which "functional signs" occurred in both benign and malignant conditions, alike. The value of these functional signs was that they focused attention to a small lesion which otherwise would have been overlooked. These lesions have been confirmed by surgery and were equally divided as to their malignant or benign nature.

Nine reproductions of roentgenograms and photomicrographs accompany this article.—*William H. Shehadi, M.D.*

Pinkerton, Philip. Psychogenic megacolon in children: The implications of bowel negativism. *Arch. Dis. Child.*, Aug., 1958, *33*, 371–380. (From: The Department of Psychological Medicine, University of Liverpool, Liverpool, England.)

The author presents a study of 30 cases designated as idiopathic or functional megacolon. He divides the megacolon into 2 different groups, (1) congenital neurogenic or aganglionic megacolon with a spastic aganglionic segment, and (2) colonic enlargement without evidence of organic lesion.

Idiopathic megacolon has been attributed to a state of chronic colonic inertia. This present investigation seeks to demonstrate, however, that megacolon can develop in children primarily on the basis of emotional factors. The condition so produced closely resembles idiopathic megacolon in clinical features and roentgenographic appearances.

All 30 cases had the common factor of conflict between parent and child in the sphere of bowel function. Parental efforts to promote bowel regularity had induced a reactive stage of negativism in the child manifested as resistance to defecation. This, in turn, led to chronic fecal retention, with ultimate dilatation of the rectum and terminal colon, if the condition was allowed to persist for a sufficient length of time. The megacolon, so produced, could not be attributed to any underlying organic lesion. On the evidence found, it is of psychogenic origin and is of common origin with the condition sometimes called idiopathic magacolon.

The conclusion drawn from the current investigation is that, while colonic inertia forms the immediate basis of the condition, its development is secondary to the state of chronic constipation which results from persistent bowel negativism.

Rational treatment should, therefore, be directed towards resolving the child's negativistic behavior, since this represents the ultimate basis of the disorder. Treatment must include exploration of the emotional factors within the parent-child relationship which determine the patient's negativistic response, and appropriate re-alignment of parental attitude, so that the state of conflict over bowel function is removed.—*James F. Martin, M.D.*

Marine, Robert, and Lattomus, W. W. Cavernous hemangioma of the gastrointestinal tract; report of a case and review. *Radiology*, June, 1958, *70*, 860–863. (Address: R. Marine, The Delaware Hospital, Wilmington, Del.)

Cavernous hemangiomata of the gastrointestinal tract are rare. Preoperative recognition is important because the condition is frequently fatal if not treated surgically. The small intestine, colon and rectum are the more common sites; the stomach and esophagus are much less common sites of involvement. Two types of cavernous hemangioma are recognized; the diffuse infiltrating type and the circumscribed or polypoid type. The diffuse infiltrating type causes hemorrhage frequently and obstruction occasionally. Phleboliths are often noted in hemangiomatous lesions in all portions of the gastrointestinal tract and can be demonstrated roentgenographically. Phleboliths are normally seen in the venous plexuses of the parametrial, vesical, and prostatic areas; when seen elsewhere, they suggest cavernous hemangioma. In patients with gastrointestinal bleeding or obstruction of obsucre origin, this point may be helpful.

A case of cavernous hemangioma associated with multiple phleboliths involving a loop of jejunum in a seven year old girl is reported. The diagnosis was made during a small bowel study. A brief review of the related literature is also presented.—*Z. Petrany, M.D.*

Varadarajan, M. G. Interposition of a loop of ileum between the dome of diaphragm and liver. *Punjab Med. J.*, April, 1958, *7*, 343–350. (From: Government Hospital for Women and Children, Egmore, Madras, India.)

The case history of a thirty-seven year old male Cooly is presented. The symptoms suggested presence of a peptic ulcer with obstruction at the pylorus, and barium meal series confirmed this. Fluoroscopic and roentgenographic examination of the chest showed a multiloculated cystic swelling between the right diaphragm and the liver. Serial roentgenograms for twenty-four hours after barium meal, barium enema study and pneumoperitoneum revealed the presence of a loop of small intestine between the liver and the right diaphragm.

Abdominal exploration via a right paramedian incision confirmed the provisional diagnosis and a fourteen inch segment of small bowel was resected. Multiple cysts were present in the loop of the terminal ileum and the pathologic diagnosis was "lymphangioma." Follow-up chest roentgenogram ten months later showed absence of the multiloculated

swelling beneath the right diaphragm.—*Richard E. Kinzer, M.D.*

Burnett, W., and Shields, R. Movements of the common bileduct in man; studies with the image intensifier. *Lancet*, Aug. 23, 1958, *2*, 387–390. (From: University of Glasgow, Glasgow, Scotland.)

The authors have studied the common bile duct in 10 patients by injecting contrast medium following cholecystectomy. Similar findings have been observed in each. By image intensification and cineroentgenography they have been able to demonstrate peristaltic movements which propel bile distally along the duct and into the duodenum. Consequently they believe that the common bile duct functions as an actively contracting duct rather than a passive transmitter of bile. This view may permit the consideration of common bileduct colic as an entity similar in nature to ureteric and intestinal colic. They feel that there is sufficient intrinsic muscle in the bile duct to produce these rhythmic movements. Respiratory and duodenal movements have also been studied and it is concluded that these play no great part in emptying the bile duct.—*Arthur E. Childe, M.D.*

Salzman, Emanuel, Watkins, David H., and Rundles, William R. Opacification of radiolucent biliary calculi. *J.A.M.A.*, Aug. 2, 1958, *167*, 1741–1743. (Address: E. Salzman, West Sixth Avenue and Cherokee Street, Denver 4, Colo.)

The authors report the case of a seventy-nine year old man in whom radiolucent biliary calculi were opacified by the daily oral administration of 3 gm. iopanoic acid (telepaque) for 4 days, 1 gm. after each meal. Roentgen examination on the fifth day revealed opacification of numerous large, irregularly shaped calculi in the distal right and left hepatic, common hepatic and common bile ducts to the ampulla of Vater, in spite of the fact that these had not been visible at cholecystography nine days before. Three weeks later the calculi had again become nonopaque, but they were re-opacified by the four-day administration of iopanoic acid. The final examination three weeks later showed that they had again become invisible.

In vitro studies showed that the opacification is the result of a deposition of iopanoic acid or an iodinated product on the surface of the stones.—*Arthur E. Childe, M.D.*

Verstraeten, J. (Gand, Belgium.) Postcholécystectomie. (Postcholecystectomy.) *J. belge de radiol.*, 1958, *41*, No. 3, 255–300.

The so-called postcholecystectomy syndrome does not actually represent a definite anatomicophysiologicopathologic entity. It is essentially a clinical condition associated with varied and complex complaints, often vague, pertaining to the retained portion of the biliary tract after cholecystectomy, namely the bile ducts.

The term implies erroneously that the symptoms have resulted from the cholecystectomy or postoperative modifications. It becomes more apparent that the pathologic changes existed for the most part prior to operation and were not recognized at the time of operation.

Fifty cases were studied, 8 males and 42 females. The ages varied between twenty-six and seventy-eight years. These were divided into 3 groups.

The technique used calls for a target film distance of 1 m. (40 inches), 68–85 kv., 30 mas. for routine work, and 100 mas. for laminagraphy.

I. The first group consisted of 22 asymptomatic individuals whose gallbladder was removed because of calculi, 3 were males and 19 females. These patients submitted for examination in good faith with no reason on their part for such a procedure. The examination was done to determine standards for: (a) the optimal time for making roentgenograms; (b) the use of "modifiers" and the best way to give evidence of their effect; (c) to determine the incidence of certain patterns and abnormalities; (d) to demonstrate that anomalies are not necessarily causes of complaints.

When the bile ducts are faintly visualized, morphine is used to close the sphincter and to bring about better visualization. This helps to differentiate organic from functional lesions. This is followed by the use of amyl nitrite, which relaxes the sphincter and brings about accelerated evacuation.

The Boyden (eggnog) meal was used in 9 out of 10 patients. This resulted in collapse or emptying of the ducts within fifteen minutes.

In laminagraphy 5 sections were made 0.5 cm. apart. For true evaluation of the condition of the biliary tract, it is best to have pre and postoperative cholangiograms for comparison.

Angulation of the ducts and deformity may be due to trauma during surgery, to sutures, or other operative interference.

In 8 of the asymptomatic patients the residual stump was 2 to 20 mm. long. The chief difficulty encountered in the examination was satisfactory visualization and evaluation of the pancreatic portion of the common bile ducts.

II. The second group consisted of 10 cases with vague symptoms, 9 females and 1 male. The average diameter of the ducts was 4–13 mm. and the findings were as follows: 4 retained cystic duct stump; 2 residual or reformed gallbladder (1 sphincteric incompetence, 1 hypertrophy of the sphincter with stenosis); and 4 nonvisualization of the distal common bile duct.

III. The third group consisted of true postcholecystectomy syndrome, the causes of which are: (a) *in-*

trinsic organic lesions: lithiasis; cystic duct stump, so-called residual or "reformed" gallbladder; postoperative stricture; stenosing odditis; and (b) *residual pancreatitis:* tumors of the biliary tract, especially near or at the ampulla; functional disorder at the level of the sphincter of Mirrizzi; and dyskinesia and dystonia of the sphincter of Oddi.

Special mention of choledochoduodenostomy is made, where the sphincter is absent, with no bile duct visualization after intravenously injected contrast medium. However, air and barium reflux, following a gastrointestinal series is observed which gives satisfactory delineation of the bile ducts.

Eighteen reproductions of roentgenograms accompany this article.—*William H. Shehadi, M.D.*

Wolf, H. G. Beiträge zur Diagnose und Ätiologie des spontanen Pneumoperitoneums beim Neugeborenen. (Contributions to the diagnosis and etiology of spontaneous pneumoperitoneum in the newborn.) *Radiol. clin.*, July, 1958, *27*, 193–197. (From: Universitäts-Kinderklinik, Wien, Austria.)

Three cases of infants with spontaneous pneumoperitoneum are reported.

Case I began vomiting on the second day of life and had no bowel movements for four days. Roentgenograms showed a large pneumoperitoneum with downward and medial displacement of the liver and spleen, suggesting a meconium ileus or congenital megacolon. At operation, a perforation of the ascending colon 4 cm. distal to the cecum was found. At autopsy, aganglionosis extended from the rectum to the splenic flexure.

Case II began vomiting on the second day. Meteorism was particularly noticed in the upper half of the abdomen. Roentgenograms showed small crescentic air pockets under the right diaphragm and in the right paravertebral region at the level of the liver. A contrast meal examination demonstrated stenosis of the jejunum and a complete obstruction at the splenic flexure. At operation, a volvulus of the transverse colon was found as well as a perforation of the upper ileum.

Case III developed subcutaneous emphysema of the neck on the third day of life. Roentgenograms showed a pneumomediastinum and pneumoperitoneum. At autopsy, beside a tetralogy of Fallot, a diffuse bronchiolitis was found. The latter was undoubtedly responsible for the pneumoperitoneum, which arose when air escaped from the mediastinum through openings in the diaphragm.

Pneumoperitoneum of the newborn is most commonly the result of perforation of the gastrointestinal tract due to atresia, congenital weakness, or ulceration. A rarer cause is diffuse bronchiolitis as cited in this paper.—*J. Zausner, M.D.*

Gynecology and Obstetrics

Stein, Irving F., Sr. The Stein-Leventhal syndrome; a curable form of sterility. *New England J. Med.*, Aug. 28, 1958, *259*, 420–423. (From: Northwestern University Medical School; and Michael Reese Hospital, Chicago, Ill.)

A form of sterility observed in females with bilateral polycystic ovaries, secondary amenorrhea and, often, hirsutism is described by the author as the Stein-Leventhal syndrome. He has treated 96 such cases in thirty years.

The sterility is cured by a small wedge resection of the ovaries and puncture of the cysts through the incision thus made.

The diagnosis is made by history, physical examination, the presence of normal urinary 17-ketosteroids and follicle stimulating hormone, and direct visualization of the enlarged ovaries. The author uses gynecography (pneumoroentgenography) for preoperative visualization of the ovaries.—*Henry J. Klos, M.D.*

Hutchinson, Donald L., Bennett, Leslie R., and Gean, David A. Isotopic localization of the placenta in placenta previa. *Surg., Gynec. & Obst.*, Sept., 1958, *107*, 370–374. (From: The Department of Obstetrics and Gynecology and Radiology, School of Medicine, University of California at Los Angeles, and the Obstetrical Service and Radioisotope Laboratory, Harbor General Hospital, Torrance, Calif.)

Thirty cases are reported in which I^{131} human serum albumin was used to locate the placental site. Nine of the cases had third trimester bleeding. There were 4 cases of placenta previa and 5 cases of premature separation of a normally implanted placenta.

Five μc of I^{131} HSA was injected intravenously and two minutes later measurements were made over the abdominal surface. The abdomen was divided into 12 areas and the standard or base count was located over the xiphoid process. The accuracy of placental localization was greatest in the lower sections of the abdomen or in the region of the lower uterine segment. The counts in the upper sections or fundal region were not as accurate due to the scattered radiation from the liver, spleen and heart. Ninety per cent of the patients with a normally implanted placenta gave readings in the lower sections between 35 per cent and 69 per cent as compared with 73 per cent to 130 per cent if the placenta was located in the lower uterine segment.

The placenta was accurately localized in all of the authors' cases when it was in the lower sections. When the placenta was located in the posterior por-

tion of the fundus and in the midline, the counts were all at base level and the localization was made by exclusion. Most posteriorly implanted placentae were detectable by an increase in radioactivity over one of the lateral margins of the uterus. It is conceivable that a low lying placenta in the midline posteriorly could be missed.

The authors believe the test to be safe, rapid and reliable.—*Charles W. Cooley, M.D.*

Genitourinary System

Mayer, Raymond F., and Mathes, Gordon L. Retrocaval ureter. *South. M. J.*, Aug., 1958, *51*, 945–950. (From: The Department of Urology, Baptist Memorial Hospital, Memphis, Tenn.)

Retrocaval ureter is not produced by migration of the kidney or ureter but by abnormality of the vascular system. The condition results from a failure of the right posterior cardinal vein to atrophy and its persistence as the adult vena cava. Normally this vein disappears and the right supracardinal vein persists as the vena cava. Occasionally both cardinal veins persist, the vena cava being double, with the ureter passing between the 2 divisions.

There are no pathognomonic symptoms of retrocaval ureter. Usually the symptoms are those of hydronephrosis with superimposed infection or calculus. Hematuria has less frequently been noted. Most patients have reached adulthood before symptoms develop as the onset of hydronephrosis is usually insidious. The diagnosis is made by pyelographic findings, the upper portion of the ureter being S-shaped with displacement medial to, or beyond, the midline at the level of the third, fourth and fifth lumbar vertebrae. There is usually some associated hydronephrosis.

Lateral or oblique views show the ureter staying close to the spine in the upper one-third rather than falling away as it normally does. Catheterization of the vena cava through the saphenous vein has been performed but venography should not be necessary in cases with typical pyelographic findings.

The possibility of a retroperitoneal or renal tumor producing medial displacement of the ureter should always be kept in mind.

The treatment is surgical. The authors report 2 cases in which the retrocaval ureter was successfully corrected by division of the renal pelvis, unwinding of the ureter and anastomosis without splinting or nephrostomy drainage. The hydronephrosis was minimal in both cases, the diagnosis being made from the medial displacement and S-shaped curvature of the upper ureter.—*Peter C. Truog, M.D.*

Coppridge, Alton J., and Ratliff, Rigdon K. Unilateral multicystic kidney disease. *J. Pediat.*, Sept., 1958, *53*, 330–335. (From: The Department of Urology, St. Joseph Mercy Hospital, Ann Arbor, Mich.)

Unilateral multicystic kidney disease is a congenital condition characterized by replacement of the normal kidney tissue by multiple cysts of various sizes held together by connective tissue. It occurs almost exclusively in infants and children, and is a relatively uncommon lesion. The authors report 2 cases, bringing the total recorded in the literature to 31.

Unilateral multicystic kidney disease is a separate entity and should not be confused with multilocular cysts of the kidney, polycystic disease, solitary renal cyst or multiple simple cysts of the kidney. In multilocular cysts of the kidney, the cysts are limited to a portion of the kidney with uninvolved normal segments. The large cysts are subdivided and partitioned.

Polycystic renal disease is almost always bilateral and hereditary. In both the infantile and adult forms of polycystic disease the renal contour and calyceal pattern are retained the the pelves and ureters are normal.

In unilateral multicystic kidney disease, a palpable mass is usually present and the pelvis of the affected kidney is not visualized on excretory pyelograms. At cystoscopy, the ureteral orifice on the affected side is often absent. The prognosis is favorable as nephrectomy results in a cure.

In most of the cases recorded in the literature and in the 2 cases reported by the authors the preoperative impression was Wilms' tumor or congenital hydronephrosis.—*J. N. Ané, M.D.*

Lewis, Samuel, and Doss, R. Douglas. Calcified hydropyonephrosis. *Radiology*, June, 1958, *70*, 866–867. (Address: S. Lewis, VA Hospital, University Drive, Pittsburgh 40, Pa.)

A case of calcified hydropyonephrosis in a sixty-nine year old diabetic male is described by the authors. In reconstructing the sequence of events in the case, the authors felt that chronic hydronephrosis had developed as a result of strictures of the ureter with consequent low-grade infection and eventual healing by calcification. The clinical course was unmodified by treatment except for the diabetes, as the patient's symptoms were never sufficient to cause him to see a physician until his terminal illness.

Accompanying roentgenographic reproductions beautifully demonstrate the calcified, lobulated cystic mass in the area of the right kidney.—*John S. Alexander, M.D.*

Henriques, C. Q. (London, England.) Osteomyelitis as a complication in urology; with special reference to the paravertebral

venous plexus. *Brit. J. Surg.*, July, 1958, *46*, 19–28.

Five cases of osteomyelitis following operations on the lower urinary tract, and one occurring spontaneously in the presence of urinary infection, are presented. The sites involved were spine alone, 3 cases; pubis and left hip, 1 case; hip alone, 1 case; and shoulder alone, 1 case.

The paravertebral venous plexus and its branches are suggested as the probable pathway by which the organisms reach the bones involved. The studies of Batson on the vertebral vein system are recounted. The author includes an extensive review of the literature on this subject.

It is noted that a strong similarity exists between these lesions and osteitis pubis.—*James J. McCort, M.D.*

PERILLIE, PASQUALE E., and CONN, HAROLD O. Acute renal failure after intravenous pyelography in plasma cell myeloma. *J.A.M.A.*, Aug. 30, 1958, *167*, 2186–2189. (Address: H. O. Conn, West Spring Street, West Haven, Conn.)

Fatal complications of intravenous pyelography are extremely rare. Pendergrass and co-workers reported only 31 deaths in more than 3,800,000 intravenous pyelographic examinations. Twenty-five of these deaths occurred immediately after intravenous injection of the contrast substance and were preceded by evidences of anaphylactoid shock, cardiac arrest, or symptoms of drug sensitivity. Three of these patients died of uremia within a few days of the examination.

The authors reviewed the literature, and there were 4 deaths occurring in cases with plasma cell myeloma following intravenous pyelography. The additional case of the authors is the fifth fatal case. None of the 5 patients with plasma cell myeloma in whom pyelography is reported to have precipitated acute renal failure exhibited any of the features of drug allergy to 3 different contrast media. The clinical and laboratory aspects of the 5 cases of pyelography-induced anuria are summarized. It is interesting to note that the diagnosis of plasma cell myeloma had not been established in any of these patients at the time pyelography was performed, although it was suspected in several.

In summary, the authors state that acute renal failure may develop immediately after intravenous pyelography in patients with plasma cell myeloma. Autopsy of these patients in whom abnormal proteinuria had been demonstrated showed plugging of the renal tubules by coagulated protein casts. The authors postulate that in the presence of Bence Jones proteinuria, the dehydration produced by fasting and purging and the increased concentration of urine caused by the abdominal compression may combine to cause precipitation of the urinary protein within the renal tubules. This internal obstructive uropathy may be prevented by avoiding intravenous pyelography in patients with plasma cell myeloma or proteinuria of undetermined cause. If acute renal failure is precipitated in these patients by simple dehydration, it is important to assure adequate fluid intake at all times.—*Peter C. Truog, M.D.*

PALUBINSKAS, A. J., and HODSON, C. J. Transintervertebral retroperitoneal gas insufflation. *Radiology*, June, 1958, *70*, 851–854. (From: Department of Diagnostic Radiology, University College Hospital, London, W.C.1, England.)

The authors report a modification of the presacral method of retroperitoneal gas insufflation which has been used successfully in 34 cases.

An essential feature of this modification is the introduction of the insufflating needle into the retrorectal areolar tissue through one of the coccygeal intervertebral spaces. The desired intervertebral space is located by means of a lateral coned-down roentgenogram of the coccyx, aided by a radiopaque skin marker at about the optimum level. With proper depth control, a shortened 20-gauge lumbar-puncture needle, attached to a 10 cc. syringe filled with physiologic saline, is advanced through the interosseus space while maintaining gentle positive pressure on the syringe plunger. Entry of the needle tip into the precoccygeal areolar tissue is marked by 2 simultaneous easily recognized end-points: a decreased resistance to the passage of the needle, and a sudden easy flow of saline from the syringe. Another lateral roentgenogram of the area is obtained for confirmation after which the remainder of the saline is injected. The latter procedure serves the multiple purpose of displacing the rectum away from the needle point, assisting in the establishment of a dissecting plane for the gas to be introduced, and compressing any small vessels that may have been opened during the insertion of the needle. The patient is now ready for the insufflation of the chosen gas medium with continuous cardiac auscultation as a safety measure.

Although the procedure is slightly more time-consuming than the usual presacral technique and requires 2 roentgenograms of the coccyx, the authors feel that this modification offers the following advantages: the needle remains firmly fixed in its position; maintaining the operators, finger in the rectum is made unnecessary; a comfortable prone position can be used; and there is easy access even in obese individuals with large buttocks.—*Edward B. Best, M.D.*

SWART, B., and SCHÄFFER, A. Symptomatologie und Diagnostik der primären retroperitonealen Tumoren. (Symptomatology and diagnosis of primary retroperitoneal tumors.)

Chirurg, Aug., 1958, *29*, 357–361. (From: Universitäts-Strahleninstitut, Universitäts-Krankenhaus, Hamburg-Eppendorf, Germany.)

The literature contains about 900 reported cases of primary retroperitoneal tumors (including 7 cystic lymphangiomata).

Macroscopically, one may distinguish *cystic* neoformations, which are (a) embryonal, either teratoid (dermoid) or from the remainders of the urogenital tract, (b) lymphatic, (c) traumatic, and (d) parasitic (Echinococcus). These are usually benign in nature, but can become dangerous by excessive growth and/or secondary infection.

The origin of *solid* tumors may be (a) fibrous (lipoma, fibroma, myxoma, with composite types and/or sarcomatous degeneration), (b) vascular (malformations or dilatations of preformed vessels including angioma, endothelioma, or perithelioma), (c) nervous (neuroblastoma, ganglioneuroma, sympathogonioma) with endocrine components (pheochromocytoma), and (d) lymphatic (lymphoma).

Tumors of the spleen, liver, pancreas, and kidney must not be included in this classification, except for Wilms' adenosarcoma (embryoma) of the kidney, because of practical and clinical circumstances, since in children the latter represents 53 per cent of all retroperitoneal tumors, while second place is held by neuroblastoma (35 per cent). There seems to be an increased incidence with advancing age; for instance, 49 per cent of retroperitoneal tumors are encountered in the forty to sixty year age group. Among the 900 cases above mentioned, about one-third were connective tissue tumors. Accurate statistics are not available, mostly because none of the series contained a significantly large number of cases; thus, the incidence of "benign" tumors is given by one author as 14 per cent, by another as 95 per cent.

Because of the size of the retroperitoneal space, the tumor must enlarge for a relatively long timespan before it will cause symptoms. There may appear first gastrointestinal complaints (fullness, regurgitation, constipation), perhaps ill-defined deep-seated abdominal pain, later direct compression phenomena, such as glycosuria (from pressure on the pancreas), icterus, esophageal varices, ascites, hydronephrosis, varicocele, and so on. In children, the apparent onset may be more acute, while in the late stages, most of them have both fever and urogenital disturbances. In adults, the most frequently encountered misdiagnoses were carcinoma of the transverse colon, uterine fibroma, gastric carcinoma, ovarian tumors, fibroma of the chest wall and pancreatitis; in children, retroperitoneal tumors were misinterpreted most often as blood dyscrasia, acute abdomen, splenomegaly, tuberculosis, mesenteric or dermoid cysts, and so on.

Among the *indirect* roentgenographic signs of retroperitoneal tumor, one finds that on the "flat plate" of the abdomen, the psoas contour appears obscured, or there is a readiolucent area somewhere in the upper abdomen (due to lessened roentgen absorption because of increased fat content). Among additional investigations, since over 60 per cent of retroperitoneal tumors are located in the vicinity of the kidneys, excretory and retrograde pyelography are most likely to reveal significant findings, but even barium meal or enema study can be of help. *Direct* visualization of the tumor requires pneumoretroperitoneum, sometimes combined with aortography and/or cavography.

In the majority of cases, one will not be able to reach beyond the preoperative diagnosis of retroperitoneal tumor. Except for the endocrinologically active pheochromocytoma, or in the presence of metastases, the suspicion of tumor in the abdomen warrants exploratory surgery. In the case of benign growths, extirpation is advisable (if possible) because of the danger of further enlargement, secondary infection, or rupture into the peritoneal cavity. This paper is illustrated with the report of a successfully resected (benign) cystic lymphangioma. In the case of inoperable malignancies, or when metastases are found, palliative irradiation is indicated, because the majority of neoplasias in the retroperitoneal space are relatively sensitive to roentgen therapy. Lymphoma, if diagnosed, contraindicates surgery, but is one of the best indications for (palliative) irradiation.—*E. R. N. Grigg, M.D.*

Nervous System

Reinhardt, K. Die Vorteile der Kombination der Abrodilmyelographie mit der Diskographie. (The advantages of combining abrodil myelography with diskography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Aug., 1958, *89*, 188–197. (Address: Warndtstrasse 42, Völklingen-Geislautern/Saar, Germany.)

Myelography, *i.e.*, instillation of contrast medium (abrodil is a water-soluble organic iodinated compound) into the spinal canal, must be done before attempting surgery for prolapsed intervertebral disks. When the herniated disk is very flattened and displaced laterally, it may not be visualized adequately by myelography. Also otherwise, diskography (injection of contrast material into the intervertebral disk) provides more information than myelography on the extent and exact site of the herniation. Conversely, myelography offers a survey of an entire area, as for instance the lumbar spine, whereas a patient cannot be expected to endure five consecutive needle punctures during one session, necessary to give comparable range of investigation.

In order to take advantage of both methods, the author selects the site of the original puncture (with

intent to perform myelography) so as to be over the interspace most likely to contain a herniated disk. This can be done on the basis of neurologic findings. Immediately after introduction of the needle, a lateral roentgenogram of the spine is taken, and if the needle tip points toward the incriminated intervertebral space, the needle is advanced (under fluoroscopic control) until it reaches about the middle of this space. After injecting the contrast medium for diskography, the needle is slowly withdrawn until it reaches the lumbar sac, at which time myelography is performed. If, at the first check, the needle is not opposite the interspace, manipulation is not recommended, because the anterior intervertebral venous plexus may be punctured. The resultant bleeding is not dangerous *per se*, and will stop spontaneously, but not before staining heavily the spinal fluid, which may lead to considerable backache, and other complications due to the large amount of fibrinous material introduced into the fluid. When properly executed, the procedure is sufficiently safe to be recommended routinely.

Three illustrative cases are briefly discussed.—*E. R. N. Grigg, M.D.*

Skeletal System

Willich, E. Das Röntgensymptom der metaphysären Aufhellungslinien im Säuglingsalter. (The roentgen sign of the translucent metaphyseal lines in the newborn.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, June, 1958, *88*, 635–649. (Address: Zentral-Röntgeninstitut der Städt. Krankenanstalten, Bremen 23, Germany.)

The translucent metaphyseal lines, bands, or zones—depending upon their width—show sharp demarcation toward the epiphyses; however, they are often unsharp and exhibit gradual transition toward the diaphysis. In most cases they appear symmetrically and favor the lower epiphysis of the femur and the proximal epiphysis of the tibia, although they may also be noted in the iliac crest and scapula. They have been called zones of "decreased density," "metaphyseal zone of rarefaction," and "juxta epiphyseal osteoporosis." With few exceptions, they are observed in newborns in the first half-year about eight to twelve days after the action of the causative factor and are an expression of disturbance of enchondral ossification, representing a basic pathologic process of ossification. (The author will discuss the histologic substrate of these changes in another paper.) These lines of radiolucency are followed within a few weeks by lines of increased density. The latter were called previously "lines of arrested growth," but the term was changed later by Caffey to "growth lines" since they were found also in cases with accelerated growth. Technically, it is extremely important to obtain roentgenograms of an extended knee with a central ray falling vertically into the joint space.

Roentgenograms of 156 newborns who had them taken for different reasons were evaluated. The material was divided into the following groups: avitaminosis, disturbances of nutrition and metabolism, premature infants and newborns who had natal or prenatal damage, syphilis of the mother with the child sero-negative and sero-positive, blood dyscrasias, malformations, particularly congenital heart disease, and acute and chronic infections.

Besides translucent metaphyseal lines, in each of these groups there were certain specific local and clinical characteristics such as: bone atrophy, marginal spurs and infractions of the brittle epiphyseal plates (Trummerfeldzone) in scurvy; cuppings of the metaphyses and involvement of the ribs in rickets, among others. It is of interest to point out the occurrence of translucent metaphyseal lines in newborns without rickets after antiricketic treatment with high doses of ergosterone and cod liver oil. They are, however, not pathognomonic for viatmin D intoxication.

About 20 to 30 per cent of all premature infants show radiolucent lines. Seventy per cent of the mothers in these cases had a history of prenatal difficulty. A disturbance three weeks before delivery was followed by radiolucent lines, whereas lines of increased density appeared if the causative factor was present three to six weeks before delivery. In congenital syphilis, a great many of the radiolucent lines were regarded previously as specific of the disease but actually they represented a nonspecific disturbance of ossification not limited to syphilis. They are also found in about two-thirds of the mothers who had antiluetic treatment during pregnancy.

Besides the known bone changes in blood dyscrasias, metaphyseal zones of increased density followed by zones of radiolucency toward the diaphysis were described by Cocchi in cases of blood incompatibility between mother and child, especially in erythroblastosis.

In osteomyelitis, the translucent metaphyseal lines are found not only in the affected extremity but frequently also in the metaphyses of other long bones which are distant from the primary focus. This occurs especially in the initial stage of osteomyelitis.

In cases of pneumonias complicated by pulmonary abscess, empyema, or pyopneumothorax, similar changes were noted.

The translucent metaphyseal lines do not allow, therefore, any specific diagnostic conclusions and they can be only utilized in context with other clinical symptoms. It is only logical to anticipate the greatest changes in the bones at sites of most intensive growth as is the case in the lower metaphyses. A certain parallelism can be construed to the so-called periostoses which represent zones of reaction at the site of growth in thickness.—*Nathan Kriss, M.D.*